

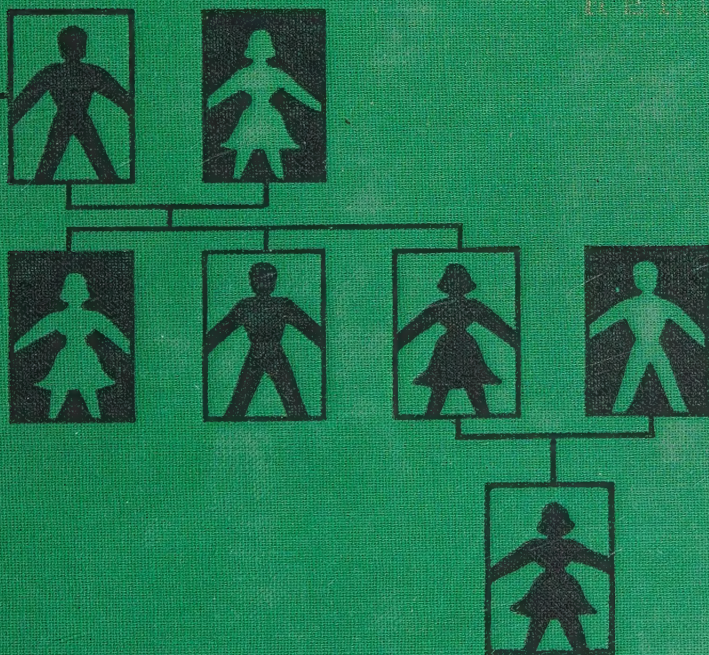
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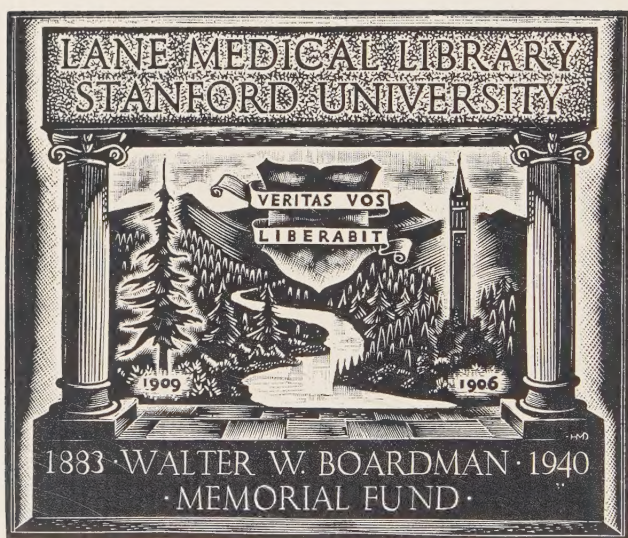
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HUMAN HEREDITY

By *Ashley Montagu*

MAN: HIS FIRST MILLION YEARS

THE CULTURED MAN

HUMAN HEREDITY

COMING INTO BEING AMONG THE AUSTRALIAN ABORIGINES

EDWARD TYSON, M.D., F.R.S. (1650-1708): AND THE RISE
OF HUMAN AND COMPARATIVE ANATOMY IN ENGLAND

MAN'S MOST DANGEROUS MYTH: THE FALLACY OF RACE

STATEMENT ON RACE

THE DIRECTION OF HUMAN DEVELOPMENT

THE NATURAL SUPERIORITY OF WOMEN

THE REPRODUCTIVE DEVELOPMENT OF THE FEMALE

ON BEING HUMAN

THE BIOSOCIAL NATURE OF MAN

DARWIN, COMPETITION AND COOPERATION

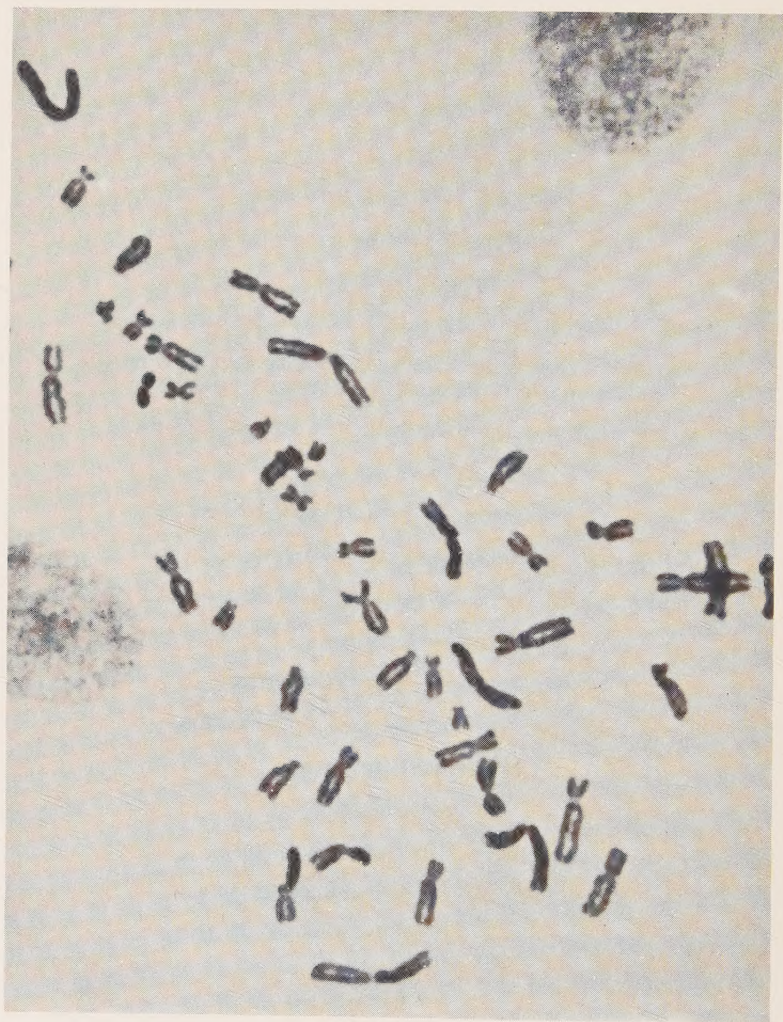
ON BEING INTELLIGENT

IMMORTALITY

EDUCATION AND HUMAN RELATIONS

ANTHROPOLOGY AND HUMAN NATURE

INTRODUCTION TO PHYSICAL ANTHROPOLOGY



Normal human chromosomes in diploid number ($2n=46$) from a newborn male's skin. Enlarged 3,250 times. (Courtesy of Dr. E. H. Y. Chu.)

Ashley - Montague, M. F.

ASHLEY MONTAGU

Human Heredity



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RLHC

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To
May Sarton

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Preface

THERE ARE several good popular books on human heredity, and the outstanding among these is Amram Scheinfeld's *The New You and Heredity*. That book is highly recommended to readers of the present volume, as is Dunn and Dobzhansky's paperback, *Heredity, Race, and Society*, and also Goldstein's *Genetics Is Easy*. The present volume, intended for the general reader, while setting out the basic facts of heredity, represents something of a departure from the conventional pattern of such books. While the usual book very properly concentrates on giving an account of the biological principles and the mechanisms involved in inheritance, the reader is all too often left with the impression that the genes and chromosomes, in their various permutations and combinations, are largely if not entirely responsible for heredity. The reader knows that there is also an environment, but somehow this seems to play merely the role of a setting within which the chromosomes and the genes pursue the even tenor of their ways. Somehow in textbooks and more popular works on genetics the environment as a factor in influencing heredity manages to get slighted. This seems to be unavoidable in works written by biologists. Their emphasis is biological, and much as they may try to avoid the biologicistic bias, they cannot help, a good deal of the time, leaving the reader with the impression that the genes and chromosomes are all. Since the facts are quite otherwise, this is an impression which requires correcting, and this I have attempted to do in this book.

Furthermore, there is a good deal of confusion about what

exactly heredity is. I have attempted to reduce this confusion, without, I hope, adding to it, or, I trust, falling into the error of extreme environmentalism as opposed to the extreme hereditarianism I desire to correct. In the development of the individual the genes are extremely important, but the environment is important too. In concentrating so much attention upon the environment I should not like to leave the reader with the impression that the genes are of secondary importance—they are not. Their importance is at least as great as is that of the environment. The power of the genes should not be underestimated—but nor should that of the environment.

I have endeavored to help the reader understand the facts of heredity clearly for himself so that he might be in a position to work out simple problems in heredity for himself. I have also provided a census of inherited disorders which should enable the reader to look up any condition in which he may be interested and determine the possible or probable manner of its transmission, and other details.

As a rule, in the pages that follow, when an unusual term is used for the first time it will be defined. Where that is not the case it will generally be defined a little later. But whenever the reader desires to check on the meaning of a term which is not immediately defined, he should turn to the glossary at the back of the book.

One of the things I regret, for which I must apologize to the reader, is that I have frequently not produced chapter and verse for many of the statements made in this book. The reason for this is that had I done so the volume would have been of considerably greater dimensions than it now is. But I wish to assure the reader that for every statement claimed as a fact in this book the evidence exists, and where I have not cited the evidence in detail, the reader may find it in the works listed in the bibliography.

To Professor Conway Zirkle of the Department of Botany, University of Pennsylvania, I owe many thanks for his kindness in reading the manuscript of this book and for his many suggestions which have served to improve it. To Professor Sheldon C. Reed of the Dight Institute for Human Genetics, University of Minnesota, I am much indebted for his helpful reading of the galleys, and for providing me with a list of counseling centers in

human genetics in the United States. To Professor Th. Dobzhansky of the Department of Zoology, Columbia University, I am grateful for his help in reading the galleys. Professor R. Alexander Brink of the University of Wisconsin kindly read my account of his discovery of paramutation, and Professor H. J. Muller read my brief account of Russian genetics, for which I herewith express my thanks. For a similar service in connection with his own and his collaborators' work I owe thanks to Dr. W. L. Russell of the Oak Ridge National Laboratory, Oak Ridge, Tennessee. Dr. Richard Levins, and Professor Max Levitan of the Department of Anatomy, Woman's Medical College of Pennsylvania, read page-proof much to the book's advantage. I thank them.

I am also most grateful to Drs. Michael Bender of the Oak Ridge National Laboratory, Oak Ridge, Tennessee, and E. H. Y. Chu of the Department of Botany, Yale University, for their kindness in providing me with photographs of human chromosomes.

To Miss Elizabeth Corddry I owe many thanks for her searching criticism of the book while it was still in manuscript, and to Mr. Jerome Fried, my editor, I am deeply obliged for his constructive assistance at every stage of the making of a book which has not been easy to produce.

This book owes its being to William Targ of The World Publishing Company, my publishers, who challenged me to write it, knowing full well that I am not a geneticist but an anthropologist. All complaints may therefore appropriately be laid at his door. I am greatly indebted to him for the suggestion, for I have enjoyed writing the book.

ASHLEY MONTAGU

Princeton, N.J.
August 1959

PART I

The Meaning of Heredity

“The teachings of genetics may be summarized in Aristotle’s saying—‘the nature of man is not what he is born as, but what he is born for’—paraphrased perhaps into the form ‘the inheritance of man is not alone what he is born with, but what he can develop.’” HERBERT S. JENNINGS, *Prometheus*, 1925

Introduction

A PART FROM identical twins have you ever seen any two persons who looked exactly alike? You haven't, and you never will. You never will because of the fact of variability—no two things are ever exactly alike—a fact that applies to all living things. That variety is the spice of life is not only true of human experience but constitutes a principle inherent in the very nature of nature. It is, indeed, the material of natural selection and the basis of social choice. For man the fact of natural variability is the best assurance against the dulling effects of uniformity and the nightmarish threats of totalitarianism.

What is the meaning of all this variation? Look about you in your own group. Observe how different people are in their appearance and in their behavior. In your own family consider how each member differs from the others, and yet how remarkably they resemble each other in some of their features and even in their mannerisms. The resemblance may be in the shape and form of the nose, the color of the eyes, body build, the walk, little mannerisms, and the like. One is very fond of music, another cannot carry a tune. One is allergic to pollens, the other is not, and so on. How do all these likenesses and differences come about in the same family and between different families? And what about the differences and likenesses among so-called "races"? Why are some people black and others white? Why do most Chinese and Japanese have "almond-shaped" eyes? Why are Pygmies pygmies? Why do people vary in intelligence so much? Which is stronger, heredity or environment? Should cousins marry? Should persons with such

and such traits marry? What are "bluebloods"? What is . . . in short, what is heredity?

The questions just asked, and others of a similar sort, are all the concern of the science of heredity, genetics—and of the special branch of that science which deals with human heredity, human genetics, which is what this book is about. Genetics is the branch of biology concerned with the manner in which inherited differences and resemblances come into being between similar organisms.

In a world in which many human beings, for one reason or another, have been caused to feel insecure, it is a common response to want to cling to one's kind and to reject anything that is different. Hence, many people tend to commit the error of concentrating on likenesses without realizing the great importance and value of differences. Differences are what make the world go round, and if it were not for differences there would be no likenesses—nothing endures but mutability. As we learn to understand the causes of difference, variation, we learn to appreciate the meaning of differences as we see them among human beings of every kind and variety; far from utilizing those differences as pegs upon which to hang our prejudices or private peevs, we begin to perceive them as points of interest which, through the greater understanding we have acquired, serve to humanize and enlarge our interest in the world in which we are living. This is a world in which there exist not only physical differences and biological differences between individuals and between groups, but also cultural differences, that is, differences in the ways of life of peoples. There are social differences, caste differences, class differences, political differences, national differences, and international differences. Somehow most of these differences have gotten themselves all mixed up in the minds of many people.

It is thought and believed by many that what a person is born with is somehow related to what he later does in life. To a certain extent this is, of course, true, but when that belief is extended to mean that what a person is born with *determines* what he will later do in life, and that therefore heredity is fate, a kind of predestination, such a belief becomes stultifying and very damaging. Fortunately it can be shown to be quite unsound. Yet there are millions of people who subscribe to the belief that if you are born a member of one "race" or ethnic group you are unlikely to achieve

as much or achieve it as well as the member of another race. There are millions of people who believe that race and society are inseparably connected, that some civilizations are highly developed and others less highly developed because the heredity of the groups involved differs in a manner significant enough to account for the differences in civilization. Millions believe that the differences in intelligence between individuals are due to heredity, and that therefore there is very little one can do about improving an individual's intelligence, which is set and determined by heredity. And then there are the plays like Clemence Dane's *A Bill of Divorcement*, which has been seen by millions of people in its stage and movie versions, its tragic theme being that because a father was insane his daughter could not marry, for fear she would pass on his insanity to her children or manifest it herself. More recently we have had William March's novel *The Bad Seed*, made into both a play and a movie, which demonstrates how the "bad" heredity of the mother, whose mother, in turn, was a murderer, shows up in her daughter, who has inherited her grandmother's disposition to engage in the not so fine art of mass murder. The novels, short stories, collective myths, and loose talk about these matters together constitute a formidable miseducative force concerning the facts of heredity. Add to this the confusion that reigns in the minds of many persons who are regarded by the public as the kind of people who *should* know, such as doctors and even biologists, and we have a quite sizable body of epidemic confusion about what heredity is and how it works. Finally, there is the word itself. It has more than once been suggested that we would be better off if the word "heredity" could be dropped altogether. And there is sound sense in the argument that the long-standing abuse of the meaning of a word constitutes the best reason for its total exclusion from common usage. Unsound words make for unsound ideas, and unsound ideas tend to result in unsound action. The word "race" is a horrid example. But most of the trouble is not with a word but with its users. And the difficulty with those who misuse "heredity" is that they simply do not give the word its full meaning, but use it rather in a too limited sense. That sense is usually far too fateful, too gloomy and unhopeful.

On the other hand, when heredity is understood in the light of the scientifically discovered facts, it is seen to be a science which yields findings that enable us to improve the welfare and general

happiness of mankind. No science is in itself either hopeful or unhelpful. A science is simply a system of knowledge derived from observation, study, and experiment calculated to determine the nature or principles of what is being studied. The truths revealed by such systematized knowledge make it possible for us as human beings to apply them to the solution of human problems. It is when we acquaint ourselves with the truths revealed by the science of heredity that we are then able to see what needs to be done and what may be done with human beings as such, what may be done with human beings as biological *and* social organisms.

Knowledge is not the end of wisdom but only the beginning of it. A knowledge of the facts of heredity should form part of the intellectual equipment of every citizen for the simple reason that the very process of being human requires an adequate knowledge of the principles of heredity if one is to be successfully human. By that I mean that knowledge of the facts of heredity is indispensably necessary if one is to have the beginnings of an understanding of the nature of human nature. Had such understanding been widespread among the German people, for example, they could not possibly have fallen for the racist theories of Hitler and his minions. If they had been able to identify those theories for the poppycock they were, they might have been able to go on from that point to recognize Hitler's political programs and prognostications for what they were. False biology and fantastic theories of heredity, often with the full consciousness of their falsity, were foisted upon a whole nation because the greater part of that nation had not been educated in the facts, an education they could easily have received during their school days, for as we shall see, there is nothing intrinsically difficult or complicated about the essential facts of heredity.

In the name of fantastic theories of heredity millions of human beings were deliberately exterminated by the Nazis. The human tragedies, the suffering, the losses to the human species as a whole thus produced can never be totaled—they add up to something beyond computation. Politics and heredity are indeed closely linked. It was Aristotle who defined politics as the complete science of human nature. Our politics will be greatly influenced by the view that we take of human nature, and often it works the other way round, our view of human nature being influenced by our politics. It has been shown, for example, that scientists work-

ing in the field of heredity tend to divide up into two camps, the environmentalists, who believe that the environment is a more powerful influence than heredity, and the hereditarians, who believe that one's biological heredity is a more powerful influence than the environment. When inquiry was made into the political beliefs of a sample of such scientists, it was discovered that the line-up was pretty much as one might have expected: the environmentalists were almost invariably Democrats, the hereditarians almost invariably Republicans. And the amusing thing was this: two of these scientists who were socialists when young were environmentalists in their socialist stage of development, but when they became older and tended toward Republicanism they were hereditarians! Dr. Nicholas Pastore has most interestingly presented these facts in his book *The Nature-Nurture Controversy*. Dr. Pastore's conclusion was that in general the political allegiances of the scientists were significantly related to their position on nature-nurture questions, and that in most cases it was probable that they were unaware of the specific impact of their political loyalties upon their scientific thinking. The opposite, however, is also true, namely, that some scientists modify their political views as a consequence of the development of their scientific ones—as was the case with several of the individuals mentioned in Dr. Pastore's book. Thus, when scientists tell us in a discussion that they are themselves uninvolved, we should take particular care to check on this and then allow for any involvement we discover in evaluating their assertions. This is a very necessary precaution, especially when discussing so important a matter as human heredity.

Scientists with political axes to grind—some of them of the highest scientific distinction—have been responsible for some most unfortunate social changes. Such scientists are likely to be anxious to introduce “reforms” in society wherever they can. As a consequence of their activities our unfortunate immigration laws were first put into the form they have had ever since, except that they have gone from bad to worse, since politicians uneducated in the facts of heredity began to modify them. Many of the sterilization laws enacted by many States of the Union were also largely the work of politically minded scientists and pseudo-scientists (see pages 289–291). The immigration laws were designed to keep out the “unfit,” and the sterilization laws were calculated to prevent

those judged to be "unfit" from reproducing their kind. In fact, both laws achieve nothing of the kind, but, in the case of the immigration laws, serve to work great injustice upon thousands of human beings who seek to become Americans and from whose admission to these shores America would greatly benefit, and, in the case of the sterilization laws, put a most dangerous and unwarranted power into the hands of the State. Politics and heredity are far from unrelated to one another.

Unquestionably there are certain individuals who should be discouraged from reproducing. But it is extremely necessary to have a thorough knowledge of the hereditary processes involved in particular cases before one may make such a judgment. Always the genetic processes involved in the transmission of traits in cases of this kind must be diagnosable. And, of course, such diagnoses will be possible only on the basis of earlier studies made on similar cases. It is one of the purposes of this book to acquaint the reader with the grounds upon which such diagnoses are made.

What we know about heredity in human beings has been largely learned through observation, though a good deal has also been learned by experiment with human beings. What we have learned in the laboratory by experiment upon lower animals we have found, upon further study and comparison, is applicable to virtually all living things, plant and animal. The same basic biological laws of heredity apply to plants, and flies, and cats and dogs, and monkeys, apes, and men. In the laboratory, using the experimenter's favorite animal, the fruit fly (*Drosophila melanogaster*), it is possible to study some 20 to 25 generations in a year, owing to the rapid development of this fly. Counting thirty years to a human generation, this means that it would take 600 to 750 years to study the heredity in the number of human generations that the fruit fly produces in only a year. We have been helped in the understanding of human heredity by many other lowly animals—the mouse, the guinea pig, and man's oldest friend, the dog. But from the standpoint of heredity man continues to be the principal object of his own interest and study, and in this book we will report not on fruit flies but on human beings . . . without forgetting our debt to the fruit flies and other animals from whom we have learned so much about ourselves.

CHAPTER 2

In the Beginning

A million million spermatozoa,
All of them alive:
Out of their cataclysm but one poor Noah
Dare hope to survive.

And among that billion minus one
Might have chanced to be
Shakespeare, another Newton, a new Donne—
But the One was Me.

Shame to have ousted your betters thus,
Taking ark while the others remained outside!
Better for all of us, froward Homunculus,
If you'd quietly died!

SO WROTE Aldous Huxley's "Fifth Philosopher." The view is somewhat extreme. There has been only one Shakespeare, and Newtons and Donnes are extremely rare. The conditions that go to make a Shakespeare, a Newton, and a Donne occur by chance, and the chances of their occurring in any generation of human beings are extremely small. So it is far better that our "froward Homunculus" (forward little man) came through, for if we were to wait for the Shakespeares, the Newtons, and the Donnes to make their appearance, the human species would have ceased to exist long ere this . . . there just aren't that many geniuses in the make-up shared by all humanity. What is that make-up?

Every human being starts off as a fertilized egg. Every female at birth already has in her ovaries well over 400,000 eggs or ova. As the female, the potential mother, grows (increases in size) and develops (increases in complexity) the ova grow and develop. Shortly after the female reaches puberty the ova begin to pass out of her ovaries, usually one at a time, approximately each

twenty-eight days, and to pass into one of the tubes or oviducts (*fallopian tubes*), the fimbriated openings or commencements of which are situated just above the ovary on each side, and thence pass into the womb (*uterus*). When the female marries and pairs with a male he introduces into her reproductive tract (at each mating) something over 200,000,000 of his germ cells (*spermatozoa*). These spermatozoa develop in his testes and are ejaculated at copulation.

After a human ovum has passed into the fallopian tube of the female it has a life of a little over twenty-four hours. After this time, if it has not been fertilized by a spermatozoön, a process which occurs in the first third of the fallopian tube (that is, nearer the ovary than the uterus), the unfertilized ovum breaks down and dies. Only one spermatozoön fertilizes the egg out of the more than 200,000,000 that have been introduced into the female reproductive tract.

The human ovum is a spherical cell approximately one-seventh of a millimeter or $\frac{1}{175}$ inch in diameter, and weighing about one 20-millionth of an ounce; in other words, it would take about 20,000,000 ova to weigh an ounce. If you want to visualize the actual size of the human ovum—and you can just barely see that single cell with the naked eye—its size is about one fourth of the diameter of the period at the end of this sentence. If you think the ovum is small, it is a giant compared with the spermatozoön. You cannot see a spermatozoön with the naked eye, you must have a microscope; for its volume is about 85,000 times smaller than the ovum. It is shaped something like a very young tadpole, with a head, neck, body, and tail, and its total length is about $\frac{1}{20}$ millimeter or $\frac{1}{500}$ inch, the tail making up the greater part of this length, the head being only $\frac{1}{200}$ millimeter or $\frac{1}{5000}$ inch in length.*

*Expressed in the customary language of science we say that the diameter of the human ovum is between 130 to 140 micra (about 0.140 millimeter) where a *micron* = 1μ or 0.001 millimeter (one thousandth of a millimeter, therefore approximately 25,400 micra in an inch), and the spermatozoön is about 0.05 to 0.06 millimeters long or about 50 to 60 micra, the head being 5 micra long and 3 micra wide, the neck 5 micra long, and the tail about 50 micra long. The spermatozoön is, in fact, by far the smallest cell in the body. Some idea of its size may be gained by considering that if we laid the sperm heads like a pavement on the top of the period at the end of this sentence it would take about 2,500 to cover it. It would only take twelve eggs to cover the same area. The big egg is immobile, but the small spermatozoön is very active and can move at the rate of about an inch in three minutes.

At the present moment there are about 2,750,000,000 human beings alive on the face of this earth. Allowing one sperm and one egg to each of them as being responsible both for their existence and their genetic heredity, we have a total of 5,500,000,000 germ cells involved, a number which could be contained in about two and a half quart milk bottles. The sperm cells would occupy the space of less than an aspirin tablet. In fact, the chromosomes, the actual bearers of the hereditary particles, the genes, within the cells of this huge number would occupy the space of less than half an aspirin tablet! Reflect upon that! All the hereditary materials, the heredity of the whole human race, of all those now living could be contained within the space of half an aspirin!

Why is the egg so much bigger than the sperm? We don't know, but it is supposed that it is bigger because it carries so much of the nutriment necessary for the fertilized egg to feed on. The sperm consists almost entirely of a *nucleus* in which the *chromosomes* carrying the hereditary particles, the *genes*, are situated. The egg consists of both a nucleus and a large quantity of more or less transparent, viscous fluid called the *cytoplasm* (Greek *cyton*, cell; *plasm*, fluid). The nucleus of the egg carries gene-bearing chromosomes. That what is contained in the nucleus, rather than in the cytoplasm, is chiefly responsible for one's genetic heredity is suggested by the fact that children resemble their mothers no more often than they resemble their fathers. Mulattoes, who are the offspring of whites and Negroes, do not resemble their mothers more than they do their fathers. The nuclei of egg and sperm are, however, strikingly similar. When these are brought together at fertilization, through the union of a male with a female and the sperm fuses with the egg, the hereditary materials are combined, and the process of development commences. From the two cells thus combined into one, two will develop, then four, then eight, then sixteen, then thirty-two, and so on until the total number of body cells at birth has been reached, a total altogether of some 200,000,000,000, and in the adult some 10,000,000,000,000, containing about 7×10^{26} atoms. It has been calculated that only about forty-four divisions are necessary to bring into being the number of cells present at birth, and only four more divisions are required to bring about the adult number. At fertilization you weighed about fifteen 10-millionths of a gram; at birth (if you were a seven-pound baby) you weighed 3,250 grams.

In the nine months from conception to birth you increased your weight 2,000,000,000 times. Adults weigh about 50,000,000,000 times as much as they weighed at fertilization. Where did all this material that makes up so much of the bulk of the individual originate? Where did enough matter come from to get the baby up to the point of birth? And where did enough come from in order to turn him into an adolescent and then an adult? In both cases the answer is the same: from the environment; from the food which the mother took in and was able to pass on in chemical form to her child in the womb, and after its birth, as nature intended, through the milk from her breasts, and subsequently through the foods which human beings obtain from their environment. So we see at the outset that environment constitutes an indispensably necessary condition of development, for without the nutriment necessary for the energizing of the developing organism there could be no development.

What is environment? Anything apart from genes that can act upon or influence the genes or the organism is environment. Cells, among other things, constitute each other's environment. Small accidental variations in the environment of the egg may have a decisive effect upon development. The complexities of the mother's body constitute the baby's environment while it is in the womb. But these are matters with which we shall deal very shortly in some detail. Let us proceed to the discussion of the nature of the bearers of the hereditary materials.

The Chromosomes and the Genes

In the nucleus of every cell there lie a number of threadlike structures, which are called *chromosomes* (Greek *chroma*, color; *soma*, body). The chromosomes are so called because they readily take up the color stain that the biologist uses in order to distinguish these bodies more clearly. In the unstained state they are clear and transparent and therefore not easy to see. In man there are forty-six chromosomes in every body cell,* and half that number, twenty-three, in the germ cell, the egg or the sperm. When egg and sperm unite they each contribute their twenty-three chromosomes so that the fertilized egg from which all the body

* Until recently it was believed that the number was forty-eight. It has now been established that there are only forty-six.

cells are derived contain the total of forty-six. Animals and plants of different kinds have different numbers of chromosomes, some, like the intestinal worm (*Ascaris megalocephala*), having as few as two and others, like certain ferns, having more than five hundred. The potato has two more chromosomes than man. Number of chromosomes does not appear to be associated with the degree of complexity of an organism. In Table I is presented a list of some representative animals and their chromosome numbers.

Since the diameter of the nucleus of the cell is not much more than a thousandth of an inch, the chromosomes contained in it are extremely small. The stretched-out chromosome (it is usually coiled in the nucleus), is between two and three thousandths of an inch long, and about a ten thousandth of an inch wide. Because chromosomes are so small their activities can be followed only with the aid of a highly powerful microscope.

It is principally through studying the behavior of the chromosomes of plants and lower animals and observing the association between differences in the behavior and structure of chromosomes and differences in the behavior of plants and animals that we have learned to understand something of the mechanism of heredity. Throughout the plant and animal kingdoms that mechanism is basically similar.

The chief importance of the chromosomes is that they afford the means by which potentialities may not only be transmitted to offspring but also the means by which the variation and change may be transmitted, for it is in the chromosomes that are lodged the physicochemical packages that are themselves the basic hereditary materials, namely, the *genes*. Recently, scientists working in the laboratory have succeeded in partially following the action of the gene both structurally and chemically. This is one of the most marvelous of scientific achievements. To know how a gene produces a hereditary change seemed to many scientists not so long ago the most difficult of all problems to solve. Today that problem has been solved for at least one human trait, and the way is now open for the solution of the manner of action of many other genes.

What is the size of a gene? It is ultramicroscopically small, and therefore no one has ever seen one. Estimates of size range between 4 or 50 millimicrons in diameter—a millimicron is one millionth of a millimeter, estimated gene size is therefore between

TABLE I
CHROMOSOME NUMBERS IN ANIMALS AND PLANTS IN THE DIPLOID NUMBER

Common Name	Scientific Name	Diploid Number (2N) of Chromosomes
Intestinal worm	<i>Ascaris megalocephala</i>	2
Small crustacean	<i>Cyclops viridis</i>	4
Fruit fly	<i>Drosophila willistoni</i>	6
Fruit fly	<i>Drosophila melanogaster</i>	8
Fruit fly	<i>Drosophila obscura</i>	10
Fruit fly	<i>Drosophila virilis</i>	12
Opossum	<i>Didelphys virginiana</i>	22
Bullfrog	<i>Rana catesbiana</i>	26
Salamander	<i>Amblystoma tigrinum</i>	28
Beetle	<i>Tirirhabda canadense</i>	30
Bat	<i>Plecotus auritus</i>	32
Lizard	<i>Anolis carolinensis</i>	34
Minnow	<i>Fundulus heteroclitus</i>	36
Cat	<i>Felis domestica</i>	38
Mouse	<i>Mus musculus</i>	40
Rat (albino)	<i>Rattus norvegicus</i>	42
Squirrel monkey	<i>Saimiri sciureus</i>	44
Red titi monkey	<i>Callicebus cupreus</i>	46
Man	<i>Homo sapiens</i>	46
Reptiles	<i>Class Reptilia</i>	48
Slow loris	<i>Nycticebus coucang</i>	50
Wood rat	<i>Neotoma floridana</i>	52
Capuchin monkey	<i>Cebus capucinus</i>	54
Tantalus monkey	<i>Cercopithecus tantalus</i>	60
Guinea pig	<i>Cavia cobaya</i>	62
Horse	<i>Equus caballus</i>	66
Herring gull	<i>Larus argentatus</i>	68
Arabian camel	<i>Camelus dromedarius</i>	70
Dog	<i>Canis familiaris</i>	78
Crayfish	<i>Cambarus virilis</i>	200
Paramecium	<i>Paramecium</i>	Estimated at several hundreds
Rhizopod	<i>Castinidium variable</i>	1,500-1,600 estimated
Plants		
Some fungi		4
Pea	<i>Pisum sativum</i>	14
Onion	<i>Allium cepa</i>	16
Primrose	<i>Primula sinensis</i>	18
Corn	<i>Zea mays</i>	20
Tomato	<i>Lycopersicon</i>	24
Wheat, cultivated	<i>Triticum vulgare</i>	42
Potato	<i>Solanum tuberosum</i>	48
Cherry	<i>Prunus laurocerocerasus</i>	170-180
Mulberry	<i>Morus nigra</i>	224-308
Fern	<i>Ophioglossum vulgatum</i>	512

$\frac{1}{250,000}$ and $\frac{1}{20,000}$ millimeter, or about one half-millionth to one forty-thousandth of the size of the period at the end of this sentence.

How many genes are there in each of the twenty-three chromosomes? Estimates range all the way from about 500 to about 5,000, and for the gene content of all twenty-three chromosomes the estimated number runs from about 10,000 to 100,000, with about 30,000 as the estimate most in favor, that is, with about 1,300 genes in each chromosome (with the exception of the male Y chromosome). These estimates are for the genes in the sex cells, the genes in the body cells having a full complement of forty-six chromosomes would have double the number of genes given in the above estimates. In the fruit fly, *Drosophila melanogaster*, in which there are four pairs of chromosomes in each cell, it has been estimated that there are between 5,000 and 10,000 genes in each cell. Man has twenty-three pairs of chromosomes, so if we tentatively award him the same number of genes that *Drosophila melanogaster* is believed to have on at least one chromosome, that is 1,250, then man would have at least 28,750 ($1,250 \times 23$) genes in the chromosomes of just his sex cells, and trillions throughout his body. In a single mating the possible combinations between the twenty-three chromosomes of the male and those of the female are 8,388,608 or 2 raised to the twenty-third power, and the chance of any one such combination being repeated more than once is 1 in approximately 70,000,000,000,000 or $2^{23} \times 2^{23}$. The different combinations which a 30,000-gene system can assume reach a stupendous figure, that is, $2^{30,000}$. This is on a purely quantitative basis. When the physicochemical factors and those of the environment are introduced as modifying agencies, the possible differences in human development within the limits of the species become practically infinite.

A gene is a giant molecule believed to consist mainly of a chemical substance called deoxyribonucleic acid or DNA for short. The stuff out of which life is made consists of protein (Greek *proteios*, prime), and nucleic acids constitute the blue-prints that direct the manufacture of proteins. DNA is so structured that it carries within itself a kind of Morse code. Each chromosome contains hundreds of genes, possibly thousands—we don't know exactly how many. In the last paragraph we settled for some 1,250 genes on each chromosome. The genes are strung

together in a row. A gene relating to a particular response or trait has a specific position or locus in the string. Nucleic acids are the blueprints which the proteins must follow if the body is to be properly built. If the blueprint isn't followed for some reason, something is bound to go wrong at the point of departure from it, and the result may be a slight or a serious disorder. The evidence strongly indicates that the nucleic acids control the making of the body's characteristic living substances—its proteins—and that DNA carries the master plans or code. The information contained in the code is there in a particular order, and this order determines the order in which amino acids (chemical compounds that are the building blocks or structural units of proteins) fall into place in the protein molecule for which it is responsible. There are about twenty different amino acids. A protein molecule may be made up of hundreds or thousands of amino acid units. It is believed that each protein owes its uniqueness to the specific sequence of its amino acids.

It was a known defective protein in man that threw the first light on the probable manner of action of genes. There is a disorder of the blood which affects mostly individuals of Negroid origin. The condition is genetically inherited, and it is called sickle-cell anemia because if you take a small sample of blood from such a person and look at it under the low power of a microscope, the red blood cells are seen in bizarre shapes frequently resembling a sickle. This is due to a difference in the chemical structure of the oxygen-carrying part of the red blood cell, the *hemoglobin* (*hema*, blood; *globus*, globe). By a most ingenious systematic chemical and electrical analysis it was found that the difference between normal hemoglobin and that which causes sickle-cell anemia is due to a difference in a single amino acid. The person afflicted with sickle-cell anemia is most unfortunate, and yet this severe disease is due to the simple fact that a glutamic acid unit has been replaced by a valine unit!

The theory of the nature of chromosome and gene is, then, that the chromosome consists of a chain of long DNA molecules and that the genes are segments of the chain. The DNA molecule, of which there are thousands in each chromosome, is conceived of as being made up of two chains that are twined around each other in a spiral structure thousands of turns long, linked together by certain basic nitrogenous substances (adenine and thymine or

guanine and cytosine). The DNA molecule has been likened to an enormously long winding staircase, with the stair treads corresponding to the cross-linking chemical bases. The various segments of these DNA molecules each contain their specific codes or blueprints for the plan to be followed in the manufacture and organization of the basic materials out of which the organism is to be developed.

It has long been clear that such a master-controlling system must be present in the chromosomes or genes since the materials that enter into the making of a living organism are so numerous and so complex that if it were left to chance only disorganization would result instead of organization. That, in fact, is precisely what the word "organism" means, an organized living entity, and moreover an organized living entity that functions to maintain organization and is opposed to all states of disorganization. Disorganization represents a breakdown in the system. Orderly and organized development is brought about as a result of the communication of the coded information contained in the living "tapes," the chains of DNA molecules. The genes are, as it were, the paragraphs in which the words or sentences are the nucleic acids (DNA). In 1958 DNA was for the first time synthesized in the laboratory by Drs. S. M. Beiser, A. Bendich, and H. Rosenkranz at Columbia University. They found that artificial DNA inhibits the natural substance's ability to cause heritable changes in pneumococci, the organisms that cause pneumonia. Some pneumococci are resistant to streptomycin. DNA taken from a resistant strain can make a sensitive strain resistant, *and this* change is subsequently inherited. Artificial DNA, however, stops the reaction. This discovery may lead to similar results in such conditions as cancer, in which the cells are thought to have an altered or abnormal DNA.

In bacteria it has been possible to transfer the DNA of one organism to the body of another bacterium, and in this way to transfer the heredity of one bacterium to another. From such experimental advances we may some day go on to achieve the correction of functioning in a defective gene by exposing the tissue cells in man to DNA derived from normal individuals, or DNA synthesized in the laboratory, and in this manner restore the individual to normal health.

From the example of sickle-cell anemia we have gained a gen-

eral idea as to how a gene probably acts. A gene may be regarded as a unit of reproduction, responsible for one or more specific steps in development. We have now to ask the question, How does a gene reproduce itself? And how can all the complexity that a human being represents (or an earthworm, for that matter) be reproduced as happens in the process so well called *reproduction*? The two questions are not quite the same, but they are very closely related.

Reproduction is just another word for *reduplication*. However, the word "reduplication" seems to describe rather more nearly the manner in which a gene reproduces itself. We have good reason to believe that a gene reproduces itself by making a duplicate or replica of itself. But how does a gene make a duplicate of itself?

The human species, *Homo sapiens* (Latin *homo*, man; *sapiens*, wise), as well as every other living species, owes its existence to the fact that living organisms are able to make copies of themselves. These copies reproduce themselves into other copies and in this way the species maintains its specific character generation after generation. We say then that there is an inbuilt specificity in the basic structure of every organism. The little girl who, when asked by her teacher, "What is it that an elephant has that no other animal has?" replied, "Little elephants," was, of course, perfectly correct in terms of genetics, although the teacher wanted an answer in terms of anatomy. The reason that elephants have little elephants and not little animals of other sorts is because the information contained in the elephant's genes is "coded" in such a manner as to produce copies of themselves. Precisely how this is done we do not know, but it is generally agreed that the process of copying probably resembles something like the process of making a mold or a template. When a sculptor wishes to make exact copies of his work, he uses his original piece of sculpture from which to make a mold, from which he can then make innumerable copies. The mold he makes is generally a negative cast, and it is from the negative casts that the positives are made. This is done by pouring the liquid material into the mold and allowing it to flow into every nook and crevice until it has hardened and a positive cast emerges.

A template is a pattern, usually in the form of a thin plate, used for testing accuracy of form in woodwork, machine parts, and the like, and in another form it is used in the mass production of all sorts of industrial and commercial products, such as the parts of

automobile bodies, and all sorts of familiar objects. It is this latter form of the template that many biologists believe represents a crude model of the living template by which the organism reproduces its molecular structure. The theory is that the DNA molecule by its very structure constitutes a perfect template. Consider the structure of the DNA molecule as shown in figure 1. It has already been stated that the DNA molecule has been likened to a long winding or spiral staircase. Let us, for diagrammatic purposes and clearer understanding, straighten it out, and unwind the staircase so that in the diagram it now resembles something like a spiral ladder with two uprights and a lot of rungs (treads). The uprights consist of sugars bound together by phosphates, and the rungs constitute the nitrogen bases, consisting of (1) adenine, (2) thymine, (3) guanine, and (4) cytosine. The rungs of the ladder always consist of one of the two pairs, 1-2, adenine-thymine, or 3-4, guanine-cytosine. The two uprights of the ladder are perfect complements of one another, and the bases 1-2 and 3-4 are always linked together in a pair on each rung, either in the order 1-2, 2-1, 3-4, or 4-3. There is, therefore, one base forming the left side of the rung and another forming the right side of the rung. Now suppose we cut the rungs clear down the middle of the ladder. We should then have two independent uprights with half a series of rungs, the left series oriented to the east, and the right series oriented to the west, and each complementing the other. Thus separated each half-ladder immediately picks up from the protoplasm in the cell the chemical substances which form the complementary uprights (sugar and phosphate) and bases, base 1 picking up base 2, base 2 picking up base 1, base 3 picking up base 4, and base 4 picking up base 3. In this manner the two separated halves of the DNA molecule become two separate identical DNA molecules.*

* Dr. R. L. Sinsheimer has recently shown that the dwarf virus, Phi X, which infects and destroys sewer bacteria consists of but a single strand of DNA wrapped in a skin of protein. The DNA is composed of one molecule made up of "beads" of nucleotides. Apparently a single strand of DNA is sufficient for the dwarf virus to reproduce itself. Twenty minutes after Phi X invades a cell it forces the cell to manufacture about 300 Phi X viruses, each capable of infecting a new cell.

Whether the dwarf virus is unique with its single strand of DNA is not at present known, but the single strand of DNA is built on the same pattern as the double one. The virus being a somewhat primitive form of life, it is not surprising that it shows the simplest form of DNA.

The process of doubling or copying thus described is at present a hypothesis. But it is a pretty good hypothesis, and there are many scientists now at work attempting to discover whether or not this is how genes, the hereditary materials, really do reproduce themselves. In any event, whatever the manner in which the process of doubling may be achieved it is believed to occur during the process when the cell is reproducing itself, a process called *cell division*. The doubling occurs long before the actual division of the cell, so that the daughter cells will have as many genes as the mother cell. During one of the phases of cell division the threadlike chromosomes are seen to duplicate themselves, so that where formerly there were a certain even number there are now double that number. It is probably at this phase of cell division that the genes are also duplicated in something like the manner described above.

Now, it should be quite clear that if chromosomes duplicate themselves, and the genes are duplicated during cell division, there must be some mechanism which holds the number constant in each body cell. Every cell in the human body contains forty-six chromosomes or twenty-three pairs. The only exception to this statement being that the germ cells contain a total of only twenty-three single or unpaired chromosomes. And this fact gives us the answer, at least in part, as to how it comes about that the number of chromosomes and genes is not in fact doubled at every cell division. It is this process of cell division that we must now discuss.

Cell Division

“Cell division” is another name for reproduction. When we speak of cell division we mean something very definite, namely, the series of continuous changes that occur during the process of duplication in which all the elements of the cell are identically reproduced.

The process of body cell division is known as *mitosis* (Greek *mitos*, thread; *-osis*, condition, hence, the condition of forming threads). The word “mitosis” refers then to the essential characteristic of the process, which is the duplication of the threadlike chromosomes. The duplicated chromosomes then separate from each other and become members of separate nuclei in separate

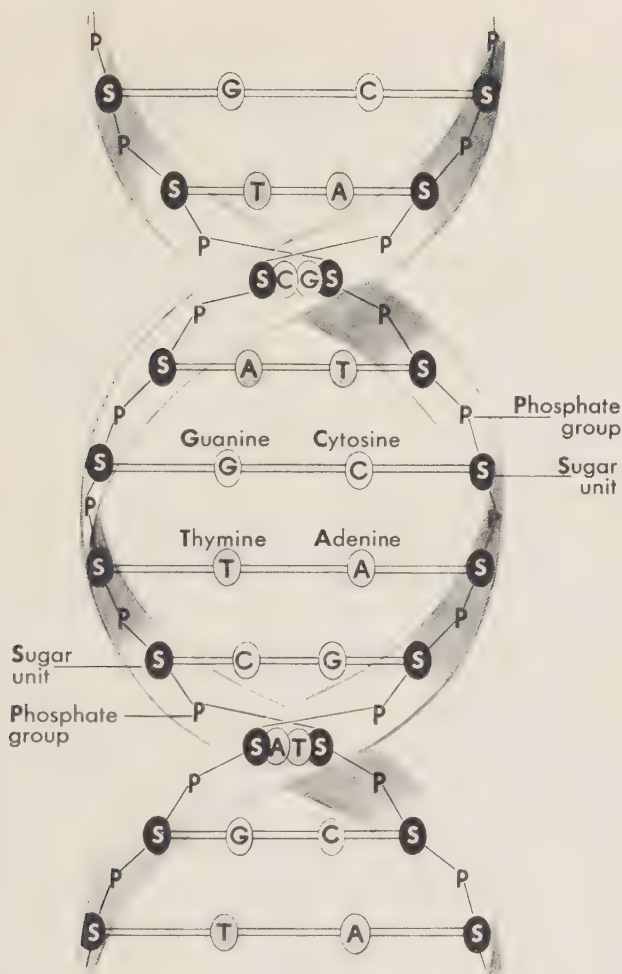


FIG. 1. Model of a Portion of the DNA Molecule. Only a few of the thousands of turns in the double spiral are shown. Each outer spiral consists of five carbon-sugar molecules *S* (deoxyribose), alternating with *P* phosphate groups—hence, a pair of sugar-phosphate (deoxyribophosphate) chains wound in a double helix. The helical chains are united by the closely fitting paired nitrogenous bases AT adenine-thymine and GC guanine-cytosine. *A*, Adenine; *C*, Cytosine; *G*, Guanine; *P*, Phosphate; *S*, Sugar; *T*, Thymine.

cells in this way: The thin, long, threadlike chromosomes remain quite separate from one another and gradually thicken into short rodlike bodies. The nuclear membrane of the nucleus within which they lie dissolves, so that the chromosomes now lie freely within the general protoplasm of the cell, the cytoplasm. A part of the protoplasm forms a spindle-shaped body which encloses the chromosomes. The duplicated chromosomes take up a position at the widest part, at the center of the spindle. The two lines of duplicated chromosomes then separate and pass in opposite directions to the tips of the spindle. Here a new nuclear membrane develops around each group of chromosomes, and the cell then divides between the two new nuclei, and there are now two complete and completely separate cells that, at least so far as their chromosomal components are concerned, are completely identical. The chromosomes then become gradually invisible, until the cell is ready to go through this whole process again. Thus, from one cell there are produced, at varying rates in a geometric progression, two cells, then four, then eight, sixteen, thirty-two, sixty-four, and so on until in the human body there are many billions of cells—the estimates range from 26,000,000,000 to more than 100,000,000,000,000.

How much time does this whole process of mitosis take? It depends upon the demands of the body. The process is often completed in less than thirty minutes. In organs that are being constantly renewed, such as the skin, the intestines, and the blood-forming tissues, cell division is rapid in order to replace cells that are being worn out and sloughed off. In kidney and liver cells the process is slower. Nerve cells are not replaced at all. Hence, damage done to cells in the brain is not reversible.

By this time it may have occurred to the reader to ask himself the question, If all the genes in all these cells are identical how then can one account for the development of the many different parts of the body? It is a good question to ask. That question happens to be one of the most difficult in the whole realm of biology. No one is at the present time in a position to answer it adequately, but it is possible to discuss the question in the light of such evidence as we have and thus suggest something of the probable nature of the answer. But before we come to that we have a little way to go yet.

Meiosis: The Reduction to Half the Number of Chromosomes in the Germ Cells

The cellular changes by means of which the full or double (*diploid*) number of chromosomes is reduced to half the number (*haploid*) in the germ cells is variously known as the maturation or the reduction division or *meiosis*, from the Greek word meaning "to make smaller." Meiosis occurs only in the reproductive cells and is characterized by *two* nuclear divisions in rapid succession.

In meiosis the chromosomes uncoil into slender threads, but instead of doubling as in mitosis each chromosome arranges itself in longitudinal contact throughout its length with the one originally derived from the opposite parent. (Remember that twenty-three chromosomes were derived from the mother and twenty-three from the father.) These two chromosomes become closely entwined about each other lengthwise (see fig. 2). In this way each gene is brought into place opposite its corresponding gene. This process is called *conjugation* or *synapsis*.

Each chromosome of a synapsed pair now doubles, just as it does in preparation for mitosis. There are now four *chromatids*, which is the name for each of the four strands of new chromosomes (the four together are called a *tetrad*). Each pair of strands or chromatids which arose in the doubling process is called a *bivalent* or *dyad*. In each group of four synapsed strands the pairs (dyads) pull away somewhat from each other. The original two synapsed chromosomes then separate, and it is found that two of the four strands (one from each dyad), in crossing one another, have broken in one or more identical places, and the broken end of each strand has united with an end of the other strand. This is a very important fact to understand, for what it means is that there has been an interchange of corresponding genes from the father and the mother (see fig. 3). By this process, called *crossing over*, genes that were originally located in a chromosome that came from the mother become part of a strand of genes that originally made up a chromosome from the father, and vice versa. As a result of crossing over, one part of a single chromosome may carry the genes for characteristics of the father, while the other

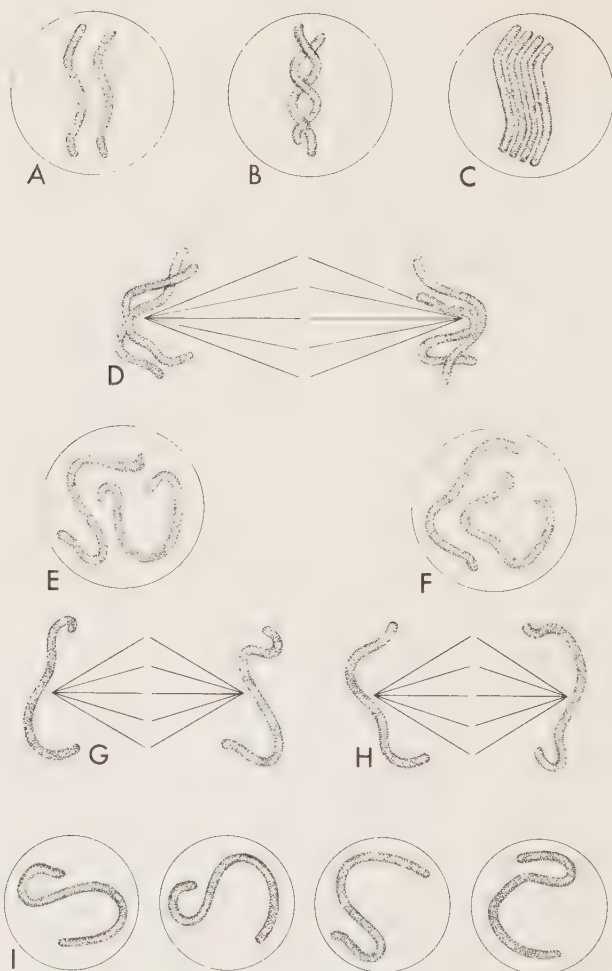


FIG. 2. Diagram of Meiosis. A. Nucleus of primary meiocyte—one pair of homologous chromosomes. B. Homologous chromosomes pairing. C. Each chromosome split and forming two chromatids. D. First Meiotic Division—chromatids separating in pairs. E and F. Nuclei of two secondary meiocytes—each with a pair of chromatids. G and H. Second Meiotic Division—chromatids of each chromatid pair separating. I. End of second division—four nuclei each with one chromatid. (After J. B. and H. D. Hill, *Genetics and Human Heredity*.)

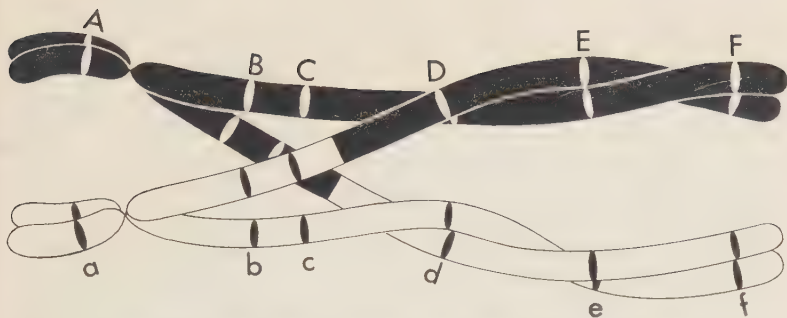


FIG. 3. Crossing Over. Here crossing over has occurred between a chromatid of the maternal chromosome, shown in white, and a chromatid of the paternal chromosome, shown in black. (After McLeish and Bryan.)

part may bear genes for characteristics associated with the mother. In this way, through our sex cells, we pass on to our offspring some of the characteristics of our own parents.

Crossing over is yet another mechanism responsible for a great deal of the kind of variation we see in individuals. We say that an individual has one grandfather's nose, but the other grandfather's forehead, while his hands are like those of one grandmother. It is partly through the crossing-over process that some of the genes and traits of both the male and female ancestors come to be represented in the individual.

But let us return to the meiotic process. Following crossing over the four strands (tetrad) become shorter and more compact, and each group of four breaks up into two pairs (dyads), each pair then separating and passing to the middle of the division spindle. This is the first meiotic division. In the second meiotic division, or *reduction division*, each pair of strands separates into two single strands, each of which passes to opposite poles. There is no duplication of the chromosomes in this reduction division. In the manner just described the double or diploid number of chromosomes is halved; or becomes haploid, in the germ cells, and the numerical constancy of the chromosomes is thus maintained in the species from generation to generation. All this is clearly set out in figure 2.

The process of meiosis results in the formation of four nuclei each carrying one chromatid from each of the four strands into which the two original chromosomes had split. But we have seen that as a result of crossing over the chromosomes may no longer

be like those derived from the maternal and paternal chromosomes. Now, since, in addition, the paired strands (dyads) arrange themselves in relation to each other in a random manner, and the crossing over can have occurred at hundreds of different places, each of the germ cells will be different from the other germ cells derived from the single original cell (mother cell) and will differ from all other germ cells derived from other mother cells. This means that every egg and every sperm carries an independent set of genes which differs from those carried in every other egg and sperm. And that is one reason why no two individuals are ever exactly alike, unless there has been no change in the structure of the pairing chromosomes in the germ cells of the parents. For the same reason—that is, because of the differences existing in the germ cells—the exact type that the parents represented can never be restored or reproduced.

And so we see how it is that by means of the process of sexual reproduction (1) variation is achieved and maintained from generation to generation, (2) characteristics are rearranged and redistributed in such a manner as to maximize the improbability of any two things ever being exactly alike, and (3) such rearrangement ensures the uniqueness of the individual.

By means of the two consecutive cell divisions in meiosis, the number of chromosomes is reduced, so that the resulting sex cells have exactly half the number of chromosomes, twenty-three, as compared with the double number of chromosomes formed by mitosis. The four cells that result from this division of one male cell are called *spermatids*. The spermatids change into *spermatozoa* by developing a head. The spermatozoön is composed almost entirely of a nucleus, a neck, and made motile by a tail.

The eggs or ova produced by the female develop in the same way. However, in females, three of the four cells resulting from meiosis, the *polar bodies* as they are called, are very small and soon disintegrate. Most of their materials have gone to form the cytoplasm of the giant mature ovum. The mature egg or sperm is called a *gamete* (marrying cell), the fertilized egg is called a *zygote* (yoked or married cell). At fertilization the double or diploid number of chromosomes is restored, and the processes of mitosis and meiosis are repeated in the formation of the body (somatic) cells on the one hand and in the formation of the germ or reproductive cells on the other.

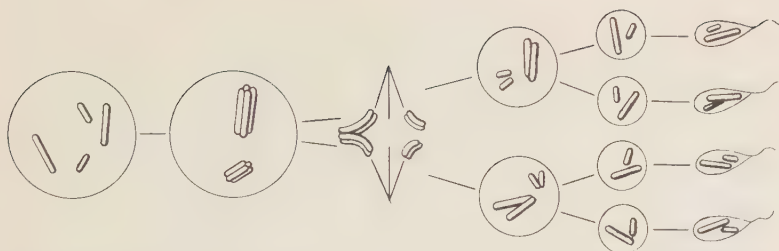


FIG. 4. The Formation of the Sperm. We commence with two pairs of chromosomes, each member of which duplicates to form a pair of tetrads or four-strand configurations. Two successive nuclear divisions result in the formation of four sperm each with one member of each pair of chromosomes.

Out of the millions of body and germ cells only a few germ cells will be directly engaged in transmitting the spark of life to the next generation. But this does not mean that the millions of other cells of the body are not involved—they are. They provide the environment for the germ cells. As Samuel Butler, the nineteenth-century thinker and writer, put it, “A hen is only an egg’s way of making another egg.”

How Sex Is Determined

At fertilization, that is, the union of the male sperm with the female ovum, the twenty-three chromosomes contributed by the sperm and the twenty-three contributed by the ovum restore the number to forty-six in the zygote. How, in what manner, is the sex determined of the organism that will eventually develop from this coming together of chromosomes? The answer is provided by the fact that out of the twenty-three chromosomes contributed by the sperm one chromosome differs in character and structure from the twenty-two others, and the same is true of the ovum. These chromosomes—which differ from the other chromosomes, or *autosomes*—are the sex chromosomes. There are two kinds of sex chromosomes: a large, well-nourished-looking sex chromosome known as the *X chromosome* and one that is a third of the size of the X known as the *Y chromosome*. Half the sperm cells carry a single Y chromosome in each of their heads; the other half carry a single X chromosome in each of their heads. Ova,

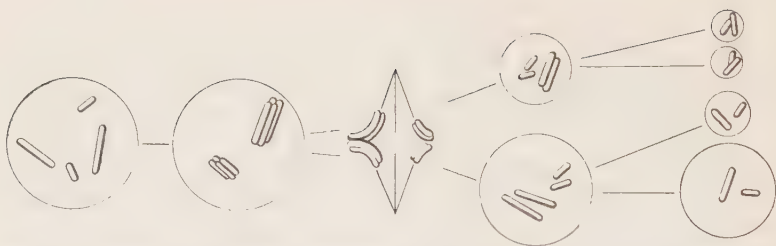


FIG. 5. Meiosis in the female results in only one functional egg—the three smaller *polar bodies* eventually disintegrate. At the first meiotic division one of the cells receives very little cytoplasm, while the other cell, the *secondary oöcyte*, receives most. At the second meiotic division the first polar body divides into two and the secondary oöcyte divides into the ovum with most of the cytoplasm and another polar body with very little.

however, all carry a single X chromosome and never carry a Y chromosome.

It is a beautifully simple arrangement. Any fertilized egg, or zygote, that results from a union of the sperm and egg will have received either an X or a Y chromosome from the sperm and always an X chromosome from an egg.

If the zygote receives a Y chromosome from the sperm, the child will be a male because it has received a Y chromosome from the sperm and an X chromosome from the ovum, and XY sex combinations always yield a male. If the zygote receives an X chromosome from the sperm, the child will always be a female, because the XX sex chromosome combination always yields a female.

From these facts it is easy to see that the sex of a child is determined “accidentally.” That is to say, it would appear that the ovum has a fifty-fifty chance of being fertilized by an X-bearing or a Y-bearing sperm and of thus producing females (XX) in 50 percent of cases and males (XY) in the other 50 percent of cases. Actually we know that more males are conceived and born than females. Between 120 and 150 males are conceived for every 100 females. We shall examine the possible reasons for this later (see pages 175–176).

Until April, 1959, it was believed that although the X and Y chromosomes are *associated* with sex they do not determine sex

in themselves, that they do not in themselves carry genes for femaleness and maleness. The Y chromosome came to be thought of as virtually empty of genes, and therefore, as having probably no influence on the determination of sex.

It was considered that sex is determined by the interaction of the genes on the twenty-two autosomes with the genes on the X chromosome. The best evidence indicated that the autosomal genes are oriented in the direction of maleness, that a single X chromosome is not sufficient to overcome this maleness orientation, but that a double dose of X chromosomes is. Hence, it was thought that the sex of the zygote depends upon the relative *number* of X chromosome genes that it receives. But that always the sex of the offspring is the result of the interaction of the autosomal genes with either one or two X chromosomes.

While these conclusions remain true for the fruit fly *Drosophila* from which creature they were extrapolated to other animals and to man, researches published in April, 1959, render it highly probable that the X and Y chromosomes play a much more significant role in the determination of sex than has been previously supposed by geneticists.

Because of the role which the X chromosome plays not only in influencing the development of sex, but also in the distribution of the genes which it carries, it has greatly helped us to understand much concerning the nature of heredity that would otherwise have remained obscure. This, again, is a matter which we shall discuss more in detail in our chapter on sex.

The Laws of Inheritance

IN AMERICA we have perhaps better opportunities of seeing how the biological laws of heredity work among human beings than in any other land—with the exception always of Hawaii. In America whites and Negroes have been mating for several centuries. Their offspring have been mating with others like themselves, backcrossing to Negroes or whites, and entering into all sorts of other conjugal combinations. Individuals belonging to the Mongoloid major group have mated with whites and with Negroes. American Indians have mated with whites, with Negroes, and with Mongoloids. In Hawaii Polynesians have formed a basic ingredient of many such intermarriages. Indeed, all the world over such bringing together of diverse heredities has been going on before our eyes, and it is to be feared we have not always understood what was going on, and only too often drawn the wrong conclusions.

Let us in a practical way, then, illustrate the basic laws of heredity as they are exhibiting themselves before our very eyes.

We have already learned that genes constitute the basic particles or packages of heredity, and we now know something of the manner in which they are transmitted to the body cells and to the germ cells. We have now to observe how those genes distribute themselves in the observable form, the *phenotype* (Greek *phainein*, to show, appear; *typos*, form; hence, the visible type), of human beings.

When a white and Negro mate, their offspring are known as mulattoes. The appearance of mulattoes, as everyone knows, is

intermediate between their white and Negro parents. Their skin color seems to be a blend of the white and the black of the parents. The hair is not as kinky as it is in the Negro parent, the nose not as flat, and so on. Such facts have given support to the ancient idea that what is inherited is a blending of two separate heredities, a mixture of the two. If such matings stopped with or at mulattoes, it would be difficult to prove that their intermediate physical traits were not due to a blending of their "bloods." But since mulattoes do go on to mate in all sorts of possible combinations, we can see at once, if we pay careful attention to the facts, that something is wrong with the blending theory, that, in fact, it won't hold up in the light of the observed facts.

When mulattoes mate, their offspring are not mulattoes. What, then, are they? Remembering that many different genes are involved and that these will be independently assorted, let us take a specific case. Two mulattoes marry, they are both the offspring of different white fathers and different Negro mothers. They have nine children. Two of these children have a black skin, kinky hair, broad noses, small ears with no lobes, and are in every other respect indistinguishable from unmixed Negroes; occasionally one or two of the children may in every respect appear to be indistinguishable from American whites; the other five or six children will show intermediate shades of skin color and other characteristics. Many other families of a similar sort that have been studied show the same kind of segregation of traits. What such studies prove is that since skin color, to deal with only one trait at a time, is undoubtedly influenced by genes, the genes do not blend with one another but retain their independence generation after generation. But the same is true of all other traits. Take, for example, the case of the mother and child in figure 6. Here is the case of a woman who is the offspring of a Basuto (South African) Negro mother and a white English father. She has brown-yellow skin, brown eyes, and kinky hair. She married a white man who had brown eyes and straight brown hair. Their two sons both had white skin, straight brown hair, and eyes like the father.

Such cases as these can only be explained on the assumption that the genes do not blend but maintain their integrity, generation after generation, and express themselves as such entities whatever the form of mating. Hence, the distribution of skin colors we encounter in the offspring of mulattoes is explicable in terms

of the fact that since mulattoes carry genes for Negroid and white traits in their chromosomes, and since these will be distributed in a random manner in the sex cells, some of the sex cells will carry mostly Negro genes, some will carry mostly white genes, and some will carry varying proportions of these genes. Now, if two germ cells which carry mostly white genes unite, the chances are great that the offspring will be mostly white in its traits. If the genes carried in the germ cells are mostly Negroid, the chances are great that the offspring will be Negroid. And if the genes carried in the conjugating germ cells are of varying varieties this will express itself in an apparent blending of traits.

In the instance of the Basuto-Negro-hybrid woman mating with a white man, the explanation here is that the woman, who was the offspring of a Negro and a white, and therefore a mulatto, carried both white and Negro genes in her ova. Her husband, who was white, carried only white genes in his sperm cells, and it so happened that his sperm united with ova which carried mostly white genes; hence, the two boys turned out to be whites like their father and grandfathers. But it might have happened that the father's sperm united with ova that carried mostly Negroid genes, in which case the boys (or girls, as they might have been) would have been less white in their traits.

Facts such as these, which were first experimentally derived from work on plants and later on animals, gave us the first of the laws of heredity.

The Law of Segregation

The first law of heredity states that in the zygote the paired genes derived from the two parents which influence the development of traits do not blend but retain their individuality and segregate unaffected by each other to pass into different gametes, and are thus able to enter into new combinations when they unite to form a new zygote. This first law (of Mendel) is known as the *law of segregation*. In man it is not possible to set up experiments to test the law of segregation, but this is possible in lower animals. Let us, therefore, illustrate the manner in which the law of segregation works on a lower animal, so that you may clearly understand it, and also so that you may clearly understand how it works in man.



FIG. 6. Segregation in Hybrid Basuto-English Mating. This woman's father was English and her mother Basuto. She married a white man. The two sons both had white skin with brown eyes like the father and straight brown hair. (After Lotsy.)

Speaking now entirely of physical traits or characters, it is known that any trait or character is under the control of pairs of genes, one gene being derived from one parent and the other gene from the other parent. Such corresponding pairs of genes are situated opposite each other on a single chromosome, and each member of such a pair is known as an *allele* (Greek *allelon*, of one another). Actually, they are not limited to acting on single characters but have many different effects and influence the devel-

opment of many different characters. Genes, then, have multiple effects. When each of an allelic pair of genes produces the same effect the genes are said to be in a *homozygous* state (Greek *homo*, same; *zygotos*, yoked). When each of the genes of an allelic pair produce different effects, they are said to be in the *heterozygous* state (Greek *hetero*, different; *zygotos*, yoked). For example, a person with two blood groups of A is homozygous for A, a person with one gene A and one gene B is heterozygous for A and B.

To trace the action of a gene we generally take a particular trait and observe how it behaves in inheritance. By this means and by experiment we can discover what gene or genes may be involved.

As a practical example let us cite an experiment on birds, retaining our interest in color, and examine what happens to the color of the feathers in such a breed as the Andalusian, a chicken similar to the leghorn. When a black bird is crossed with a splashed white, all the offspring in the first filial generation (F_1) are blue (actually they are gray, but the bird fanciers prefer to call them blue). When bred together the blues produce in the second filial generation (F_2) one fourth black, which breed true, one half blue, which breed like the F_1 blues, and one fourth white, which breed true (see fig. 7). So we have in the second filial generation one black, two blues, and one white, the blacks when bred with each other yielding only blacks, the whites when bred with each other yielding only whites, and the blues when bred with each other yielding one black, two blues, and one white. The second filial generation is more variable than the first filial generation. The blacks and whites are in equal numbers, the blues twice as numerous as either blacks or whites, the ratio being 1:2:1.

As in the case of human skin color such facts tell us that throughout the process of gene exchange and transmission the genes have not altered one bit but have retained their identity. In the first filial generation (corresponding to our mulattoes) there appears to have been blending since all the offspring are blue. But in the second filial generation there is a segregating out of the original colors as well as the appearance of the blended forms. Half are the original colors and half are blended. But the blends are not anything other than color blends; the genes remain unblended in these animals, which is proven by the fact that when they are mated their genes combine to produce pure blacks, pure whites, and blues, and the interbreeding blacks always yield blacks and the interbreeding whites always yield whites.

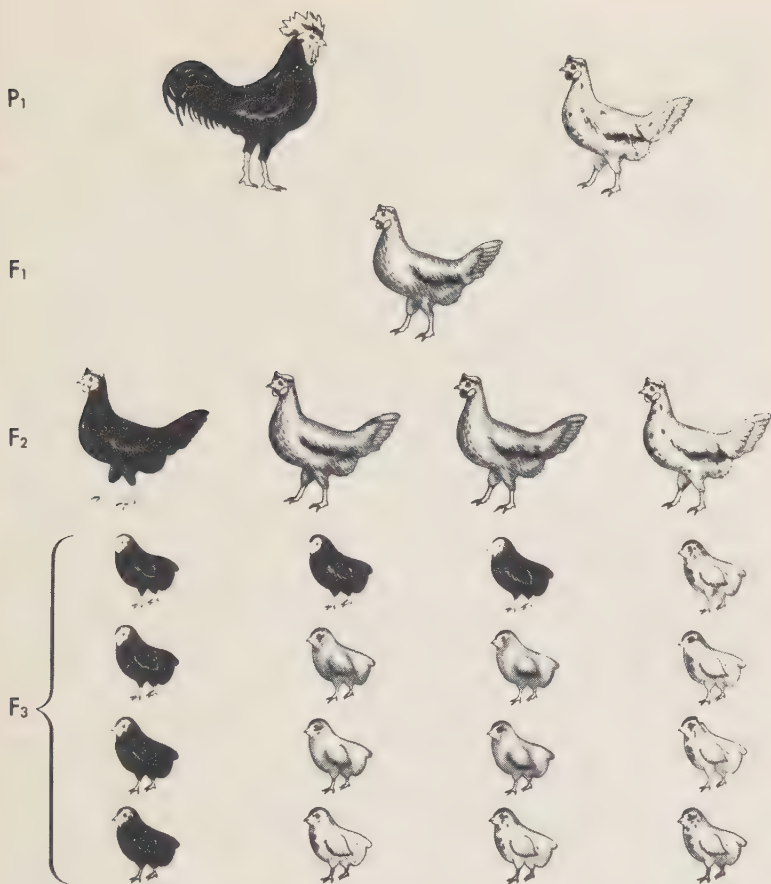


FIG. 7. Andalusian Fowl. Crossing of black and splashed-white parents results in blue birds in the first filial generation. The mating of the blue fowl produces $\frac{1}{4}$ black, $\frac{1}{4}$ splashed white, and $\frac{1}{2}$ blue in the second filial generation. In the next generation, the pattern of the mating of the blue fowl is continued; the white and black fowl breed true when mated with birds of the same color. (After Sinnott, Dunn, and Dobzhansky, *Principles of Genetics*.)

The black Andalusians were produced, therefore, by one black gene from one parent and another black gene from the other parent. The white Andalusians were produced by a white gene from one parent and a white gene from the other parent, and the blue

Andalusians were produced by a white gene from one parent and a black gene from the other. The parents contribute equally to the heredity of their offspring, and it doesn't matter whether it is the father or the mother who is one color or the other. This being so it should be clear that when the blues are mated, and they have offspring that are black, blue, and white, the genes carried by the blues must have remained distinct and must have separated from one another so that each gene entered different germ cells. Since in mating the chances are equal that each germ cell will unite with its own or with the other type—that is, black with black, white with white, black with white, and white with black—we may expect the ratio of color varieties we actually get, namely, one black, two blues, and one white. The black birds are produced by a pair of like genes, and so are the white birds—they are each homozygous for their particular color genes—but the blue birds are produced by a pair of unlike alleles, and are therefore heterozygous for the genes influencing the color of their plumage. It should be clear why blues can never breed true.

We conclude from these facts, then, that the genes influencing the development of characters are present in pairs (alleles), that each allele (member of the pair of genes) separates from its fellow allele during meiosis and enters a separate germ cell, which is the process of segregation. At fertilization the separated genes are brought together in a manner that is quite random, so that genes that find themselves paired do so by pure chance. Throughout this process the genes retain their identity.

Literally millions of observations that have been made on plants and animals support in every detail the facts resumed by the law of segregation.

This law, and the other fundamental laws of heredity, were discovered by an Austrian monk, Father Gregor Mendel (1822–1884), experimenting in his spare time in the Augustinian monastery garden at Brunn, of which he eventually became the abbot. After eight years of experiment and analysis Mendel presented his findings before the local Brunn scientific society in 1865, and published his results a year later. But the value of his work went completely unrecognized until sixteen years after his death. In the year 1900, three different scientists, Hugo De Vries of Holland, Karl Correns of Germany, and Erich Tschermak of Austria, almost simultaneously brought the importance of his work to the

attention of the scientific world. Mendel simply entitled his paper "Experiments in Plant Hybridization." He did not announce it for what it was, namely, the discovery of the age-old mystery of the laws governing the inheritance of traits. Had he done so it is just possible that he would have gained the attention for his work which was denied it for thirty-five years. "My time will come" were Mendel's last words. It has. Mendel would be astonished and delighted to know how important the science he created—*genetics*, the science of heredity—has indeed become.

What Mendel did was not what his predecessors had done. Instead of studying, as they had done, whole complexes of traits and lumping together parents, offspring, and their descendants, Mendel clearly saw that it was the inheritance of separate traits that must be studied, and that each of the members in each generation must be examined and recorded separately and as a member of its distinctive generation. Wrote Mendel in his original paper, commenting on the failure of previous investigators to do this, "It requires indeed some courage to undertake a labor of such far-reaching extent; this appears, however, to be the only right way by which we can finally reach the solution of a question the importance of which cannot be overestimated in connection with the history of the evolution of organic forms."

The Law of Independent Assortment

In reporting the results of his eight years of experiments on the garden pea (*Pisum*) Mendel "left to the friendly decision of the reader . . . whether the plan upon which the separate experiments were conducted and carried out was the best suited to attain the desired end." The friendly readers, and we know that there were more than one, apparently entirely failed to see the point.

Mendel studied seven pairs of characters in peas. These were seed form, color of seeds with coat, color of seeds without coat, form of ripe pods, color of unripe pods, position of flowers, length of stem.

Studying the offspring of a cross between a pea plant possessing round yellow seeds with one characterized by wrinkled green seeds, he found that the plants of the F_1 generation all had round yellow seeds. When the F_1 hybrids were crossed, the F_2 generation showed the two original combinations of characters, round-yel-

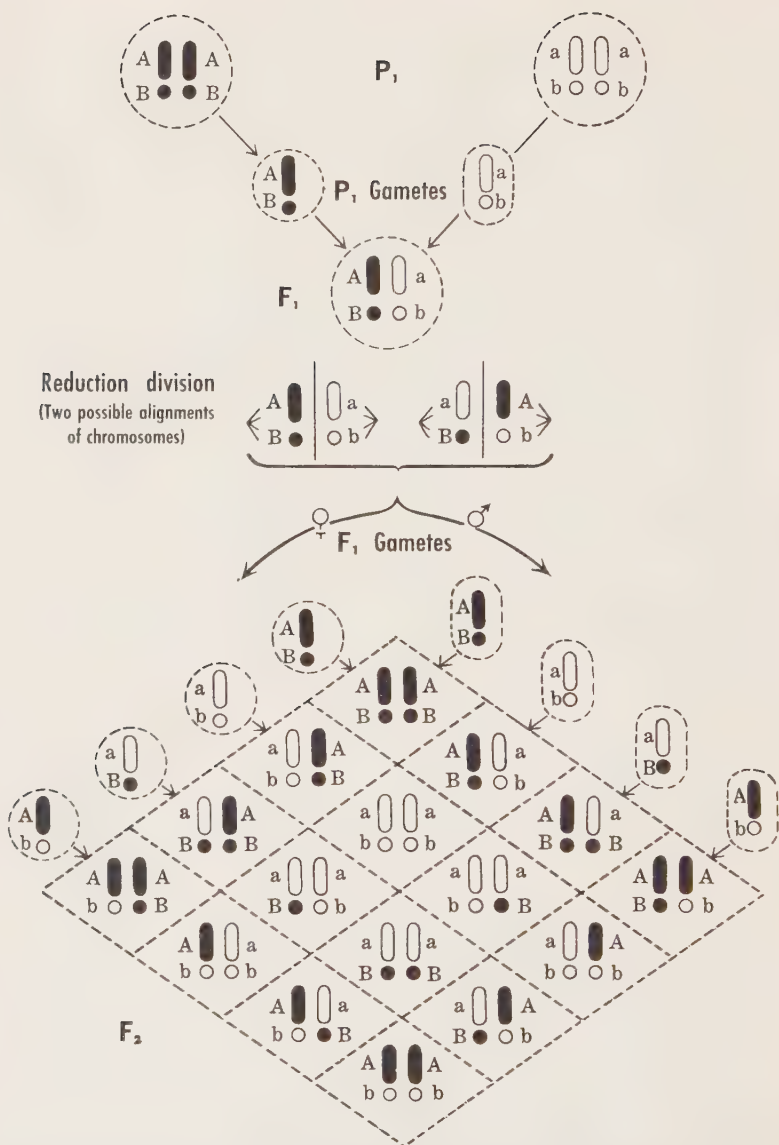


FIG. 8. Independent Assortment of Two Pairs of Chromosomes. (After Sinnott, Dunn, and Dobzhansky, *Principles of Genetics*.)

low and wrinkled-green, but also two new combinations, namely, round-green and wrinkled-yellow, in the proportions of $\frac{9}{16}$ round-yellow, $\frac{3}{16}$ round-green, $\frac{3}{16}$ wrinkled-yellow, and $\frac{1}{16}$ wrinkled-green.

By this experiment (and others) Mendel discovered the two additional laws of heredity. The second law is that the pairs of alleles conditioning the different pairs of characters in the offspring are distributed independently of each other in the way they recombine in the formation of characters. This is the *law of independent assortment*, or the *law of free recombination*. New combinations as well as old ones of genes and traits will occur, and it will be seen that every trait is inherited independently of every other trait.

The Law of Dominance and Recession

In Mendel's experiment described above, the cross between round-yellow and wrinkled-green seeds, yielded a first filial generation that in every case was round-yellow. Yet when these hybrids were crossed, the original combination of characters plus two new combinations showed up in the F_2 generation in the combinations $\frac{9}{16}$ round-yellow, $\frac{3}{16}$ round-green, $\frac{3}{16}$ wrinkled-yellow, and $\frac{1}{16}$ wrinkled-green. Evidently the two color factors differed markedly in their capacity to express themselves. One seemed to dominate the other. The same was true of the texture factors. Factors or characters that dominate over others are called *dominants*, and the characters over which they prevail are called *recessives*. Thus in Mendel's experiments with peas he found round-yellow to be dominant and wrinkled-green (which remained unexpressed in the hybrid or F_1 generation) to be recessive.

Mendel's third law states that every character is represented by two genes, one derived from each parent. When these genes are different one may dominate over or cover up the other. This is the *law of dominance and recession*.

Mendel's laws apply to all plants and animals. With these laws at their disposal students of heredity had in their hands for the first time the tools by which they have been able to explore the workings of heredity. The result is a vast and complex body of knowledge which has grown up during the last sixty years.

Until the beginning of 1959 the individuality of the gene was considered to be largely unalterable. It was believed that except

for spontaneously originating factors and such artificial factors as radiation, formaldehyde, mustard gas, colchicine, and the like, which alone were capable of producing mutations, genes remained constant and unchanged, transmitting their information unaltered from generation to generation. In January, 1959, The National Science Foundation reported the findings of Professor Alexander Brink of the University of Wisconsin which challenge the genetic axiom, hitherto prevailing, of the unalterability of the gene.

Brink found that the gene that produces color in the kernel of the purple variety of Indian corn can be permanently modified by bringing it into combination in a hybrid with the color gene from the stippled variety of Indian corn. The stippled gene produces the same pigment, but not as much, as the purple corn gene.

In crossbreeding the stippled with the purple corn variety, the stippled color gene, or an element closely associated with it, produces a heritable change in the purple color gene. When the latter is removed by outcrossing from association with the stippled gene, the purple gene is found permanently to have lost its capacity to produce purple seed pigment in normal amounts.

Purple pigmentation is dominant, so that when stippled is crossed with purple corn all the hybrids (F_1) are purple, though they carry both stippled and purple genes. Upon crossing the hybrid between purple and stippled with a white, or colorless, variety of corn, it was found that instead of half the offspring being purple and half being stippled, the half that should have been purple were now almost wholly white, retaining only the faintest quantity of pigment, whereas stippled remained unchanged.

When a similar test is made of a cross between colorless with normal purple corn which has not been previously associated with the stippled color gene in a hybrid, the conventional result is observed, namely, half the offspring are white and half purple.

The R^r gene, conditioning color, is stable in homozygous condition, but is invariably changed to a weakly pigmenting form in the progeny of heterozygotes carrying the stippled allele R^{st} . Something in R^{st} brings about a permanent change in R^r , but R^{st} is not affected by R^r in the heterozygotes. R is now changed to a new allele termed $Rr:st$. Test crosses on plants ($r^r r^r$ or $r^K r^K$) with colorless seed granules (aleurone) showed that $Rr:st$ will regularly revert partially, but not completely, toward the normal pigment-

producing level of standard R^r . Thus $R^{r:st}$ is not entirely stable, and since it does not occur at random, it cannot be regarded as a mutation. Professor Brink has suggested the term *paramutation*, to describe the fact that the phenomenon is distinct from, but not unlike, mutation.

Paramutation implies an extraordinary reactivity at some stage of cellular development of some genetic component of a chromosome when the latter is present in the nucleus with the modifying chromosome.

Professor Brink's important discovery will almost certainly be followed by corroborative findings on other forms. Meanwhile our conception of the means by which genetic variability is produced has been deepened and enlarged.

The question may be asked, What relation does Professor Brink's discovery have to the claims of the Soviet geneticist T. D. Lysenko that environmental influences such as soil, nutrition, and climate can change the hereditary properties of the gene? The answer to that question is that there is no relation between Professor Brink's discovery and that of Lysenko's claims, for what Brink's discovery proves is that a specific gene can alter the hereditary properties of another specific gene. The change is a purely genetic one, and is not an effect of external or environmental factors. (For a discussion of Russian genetics, see Appendix E.)

All this is by way of preliminary preparation for what is to follow, after we have considered some of the erroneous ways in which many people still think of the nature of heredity today.

“Blood Is Thicker than Water”

“**B**LOOD IS THICKER than water.” So it is. Blood is also stickier than water, and nowhere nearly as clear—which is exactly what may be said about a good deal of the thinking which has been left over in our society from pre-genetics days. The very phrase “blood is thicker than water” is a relic of the days when the blood was thought to be the carrier of heredity. Many people still think this is so. They believe that at conception the blood of the mother mixes with that of the father, and it is in this way that the offspring come to exhibit a mixture of the traits of both parents.

How the idea came about that blood was the carrier of the hereditary traits that were transmitted to one’s descendants is readily understandable. How ancient the idea is no one knows, but it must be very old. The fact that blood is closely related to survival is a conclusion that must have been drawn quite early in the history of man. The weakening effect or demise of the individual following an appreciable loss of blood has led all peoples of whom we have knowledge to identify blood with the vital principle itself. Among most peoples blood is regarded as a richly powerful element endowed with strength-conferring qualities as well as many others of every conceivable kind. To exchange blood or to drink blood from a common source has, among many peoples, meant the establishment of an unbreakable bond between them, for in this way they have become “blood brothers.”

If the blood contains the life force, it was probably reasoned,

then it must also contain the materials out of which the human being is built up and through which life is continued. Through such a path of reasoning, blood became the element through which not only the life-giving but the hereditary qualities were transmitted. In this way all persons of the same family stock came to be regarded as of the same "blood."

The idea of "blood" as the carrier of the heritable qualities of the "race," or nation, has led to its application in such extended meanings as are implied in terms such as "blue blood," "blood royal," "pureblood," "full blood," "half blood," "good blood," "blood tie," "blood relationship," and "consanguinity." "Blood" comes to be identified with "race," with nation, culture, and even with religion, as in the terms "Negro blood," "German blood," "English blood," "Jewish blood," and "Islamic blood."

When we examine the usage of these terms we begin to understand their meaning a little more clearly.

The term "blue blood," for example, refers to a presumed special kind of blood which was supposed to flow in the veins of members of ancient and aristocratic families. "Blue blood" is a translation from the Spanish *sangre azul*, the blood that was attributed to some of the oldest and proudest families of Castile, who claimed never to have been contaminated by "foreign blood." Many of these families were of fair complexion among a population that was dominantly brunet. In members of these exclusive families the veins would appear strikingly blue in comparison with those of the rest of the population. Hence, the difference between an aristocrat and a commoner could easily be recognized as a difference in "blood"; one was a "blue blood," and the other was not.

There is, in fact, no such thing as blue blood. The blood in the veins is dark red in color, while the veins themselves are creamy-white. The fact that the veins when seen through the skin appear to be blue is due to the refractive properties of the tissues through which they are seen, much as in the case of "blue" eyes.

The expression "blood royal," or "royal blood," refers to the widespread notion that only persons of royal ancestry have the "blood of kings" flowing in their veins. No person, it is held, however noble his ancestry, can be of the "blood royal" unless he has the blood of kings flowing in him. Kings have usually been held to belong to a special class of mankind principally by virtue of the

supposed unique qualities of their blood. In order to keep the "blood" of the royal house pure, marriages were arranged exclusively between those who were of the "royal blood." Even in ancient Egyptian times the Egyptian kings had so hypertrophied a sense of their own quality that in order not to adulterate their "blood" in any way they married their own sisters. Cleopatra, for example, was the wife of her brother Ptolemy XII, and after his early death she married her still younger brother Ptolemy XIII. As a queen, and as the Pharaoh's daughter and therefore of divine origin, Cleopatra could not possibly have married any one of a "blood" lower than her own family's.

In the manner in which they are usually used, terms like "full blood," or "pureblood," and "half blood" clearly illustrate the supposed hereditary character of the blood and the manner in which, by simple arithmetical division, it may be diluted. In this way "full blood" and "pureblood" are expressions alleged to define the supposed fact that a person is of unadulterated blood; that is, he is a person whose ancestors have undergone no admixture of "blood" with persons of "blood" considered undesirable. Within the last century these terms have come to be applied almost exclusively to persons who are not of the so-called "white race," to persons, in short, whose place is alleged to be on the lower rungs of the "racial" ladder. It is possible that this restricted usage has been determined by the fact that these expressions have generally done most service in the description of native peoples or of slaves, as in "full-blooded Negro," "pure-blooded Indian," or merely "full blood," or "pureblood." Such an imputed lowly association would be sufficient to procure the nonapplication of the term to any member of the self-styled "superior races."

A "half blood," in contradistinction to a "full blood," or "pureblood," is supposed to be half of one "race" and half of another—for example, the offspring of an Indian and a white. What is actually implied is that while a full blood may claim relationship through both parents, a half blood may claim relationship through one parent only. For example, a mulatto, the offspring of a white and a Negro, is for all practical purposes classed with the group to which the Negro parent belongs, and his white ancestry is ignored. In practice, it often works out that the half blood is not fully accepted by either of the parental groups, because of his "adulterated blood," and he becomes in the true sense of the ex-

pression a “half-caste,” belonging to neither caste, for in the Western world the so-called different “races” are treated as if they belonged to different castes.

A person is said to be of “good” or “gentle” blood if he is of “noble birth” or of “good family.” Here the assumed biological determinance of social status by blood is clearly exhibited; that is to say, a person’s rank in society is assumed to be determined by his “blood,” when, in fact, it is the other way round, “blood” actually being determined by rank. The ancestors of all noblemen were once commoners, plebians. It was not a sudden metamorphosis in the composition of their blood that caused them to become noble; it was rather an elevation in social status which endowed them with supposedly superior qualities. Such qualities are not biologically superior in any sense whatever but rather belong purely to the ascriptive variety of things; that is to say, they have no real, but possess a purely imagined existence.

The statement that a person is of “bad blood,” in the sense that he is of common or inferior character or status, is rarely encountered, for the reason, presumably, that those who use such terms have not considered the “blood” of such persons worth mentioning at all. For example, while there is an entry in the great Oxford English Dictionary for “blood worth mention,” there is none for “blood *not* worth mention.” In the sense in which blood is considered as the seat of the emotions, “bad blood” is taken to be the physiological or serological equivalent of ill feeling. In this sense, of course, “bad blood” may be created between persons of “good blood.”

The terms “blood relationship” and its Anglicized Latin equivalent “consanguinity,” meaning the condition of being of the same “blood” or relationship, by descent from a common ancestor, enshrine the belief that all biological relationships are reflected in, and are to a large extent determined by, the character of the blood. This venerable error, along with others, requires correction.

This brief examination of the ways in which “blood” is used and understood in the English language, and in Western civilization in general, renders it sufficiently clear that most people believe blood to be the equivalent of heredity, and that blood, therefore, is that part of the organism that determines the quality of the person. By extension it is also generally believed that the

social as well as the biological status of the person is determined by the kind of blood he has inherited.

These beliefs concerning blood are probably among the oldest surviving from the earliest days of mankind. Certainly they are found to be almost universally distributed among the peoples of the earth in much the same forms, and their antiquity is sufficiently attested to by the fact that in the graves of prehistoric men red pigments are frequently found in association with the remains. These pigments, authorities believe, were probably used much as they are used among nonliterate peoples of the recent past and of today, namely, to represent the blood as the symbol of life and of humanity, a belief embalmed in the expression "he is flesh and blood" to signify humanity as opposed to deity or disembodied spirit. There in the grave was the flesh, and the pigment was introduced to represent the blood.

As an example of a myth grown hoary with the ages for which there is not the slightest justification in scientific fact, the popular conception of "blood" is outstanding. It is a myth which enshrines so many errors that on that ground alone it would be desirable to outlaw this usage of the term, for it is false and extremely misleading. But what is far worse, these beliefs in "blood" are harmful in their effects and are capable of being severely damaging to human beings; hence, it is today more than ever necessary to set out the facts as science has come to know them.

In the first place it is necessary to say that blood is in no way connected with the transmission of hereditary characters, or the bases for these. The transmitters of hereditary materials, out of which characters are developed, are the genes which lie in the chromosomes of the germ cells, the ova of the mother and the spermatozoa of the father, and nothing else. The genes, carried in the chromosomes and cell substance of a single ovum and a single spermatozoön, are the only parts of the organism that transmit the genetic materials which permit the development of the organism's characters. Blood has nothing whatever to do with the transmission of heredity.

The belief that the blood of the pregnant mother is transmitted to the child in the womb, and thus becomes a part of the child, is ancient and wholly erroneous. Blood does not pass from the mother to the fetus. The blood cells of the mother are far too large (0.007 millimeter or 0.0003 inch in diameter) to be able to

pass through the placenta, and so are the blood cells of the fetus. The fetus manufactures its own blood, and the character of its various blood cells, both structurally and functionally, is demonstrably different from that of either of its parents. These facts should forever dispose of the ancient notion, which is so characteristically found among nonliterate peoples, that the blood of the mother is continuous with that of the child. Even so astute a thinker as Aristotle subscribed to this belief, stating in his work on *The Generation of Animals* (i.20) that the monthly periods, which fail to appear during pregnancy, contribute to the formation of the child's body. Modern scientific investigations have demonstrated that this and similar notions are quite false, and have completely disposed of the idea of a blood tie between any two persons, whether they be mother and child or even identical twins. Hence, any claims to kinship based on the tie of blood can have no scientific foundation of any kind. Nor can claims of group consciousness based on blood be anything but fictitious or at most a manner of speaking that is best avoided, since the character of the blood of all human beings is determined not by their membership in any group or nation but by the fact that they are human beings.

The blood of all human beings is in every respect the same, except for variations in the frequencies with which the blood group factors, the Rh factors, and the sickle-cell factors occur. But these blood groups, the Rh factors, and the sickle-cell factors are again matters of genes which happen to be differently distributed in different populations. This distribution is not a matter of quality but of quantity. There are some ethnic groups and some local populations in which certain chemical components of the blood are present that are absent in other groups, but these differences are themselves expressions of the genes which are differentially carried by such groups and populations. These differences in the chemical composition of the blood are in no way the carriers of hereditary traits themselves.

The facts about blood did not in the least serve to deter political propagandists such as the German Nazis from using the blood myths in order to set human beings against one another. The chief falsifier of the facts employed by Hitler to deceive millions of Germans and poison their minds was one Alfred Rosenberg, who was subsequently hanged by the Allies as a war criminal.

He was the author, among other works, of a book entitled *Blood and Honor* (1934), and as an illustration of the nonsense that millions of Germans took seriously, we quote him:

A nation is constituted by the predominance of a definite character formed by its blood, language, geographical environment, and the sense of a united political destiny. These last constituents are not, however, definitive; the decisive element in a nation is its blood. In the first awakening of a people, great poets and heroes disclose themselves to us as the incorporation of the eternal values of a particular blood soul. I believe that this recognition of the profound significance of blood is now mysteriously encircling our planet, irresistibly gripping one nation after another.

Nonsense such as this led to the murder of millions of human beings. That is perhaps one reason why it is important to understand the facts about the falsities involved in the myths about blood and heredity, for, as Voltaire remarked, those who believe in absurdities will be in danger of committing atrocities. Few of us are guiltless in this respect. The extravagant and preposterous claims that the Nazis made on the basis of the blood myth are paralleled by the superstitions which prevail among many other peoples. America is no exception. During World War II some of these superstitions were brought noisily out into the open when the Red Cross segregated the blood of Negroes for the purposes of transfusion. At that time the myth of "blood" seemed almost as strongly entrenched in the United States as it was among the Nazis.

The remarkable thing about the objection to Negro blood is not so much that it is based upon a misconception but that the same person who refuses to accept Negro blood may be willing to have his children suckled by a Negro wet nurse just as he had himself been as an infant. The same person will be ready to submit to an injection of serum derived from a monkey, a horse, a cow, or some other animal, and while he himself may have been suckled by a Negro wet nurse, and may even entertain the greatest affection for Negroes, he will violently object to any "pollution" of his blood by the injection of Negro blood into his own blood stream.

This is a false belief, a superstition for which there is no ground in fact. In reality the blood of the Negro is similar to that of all other human beings. For purposes of transfusion, or any other purposes, it is as good as any other blood.

The objection to Negro blood is, of course, based on the antique misconception that the blood is the carrier of the hereditary characters, and since the Negro is regarded—quite erroneously—as possessing “racially” inferior characters, it is feared that these may be transmitted to the receiver of the transfusion. Both ideas are unsound.

Telegony

Left over from the days when heredity was regarded as something that was transmitted in the “blood” is the widely diffused belief that once a female has mated with a male, no matter how many other males she subsequently mates with, the heredity of her offspring by these later males will all be affected by the heredity of the first male with whom she originally mated. This belief in the supposed transmission of the hereditary characteristics of one sire to the offspring subsequently borne to other sires by the same female is known as *telegony* (Greek. *tele*, far, distant; *gonia*, progeny). Thus, it was believed, a pedigreed horse having once mated with a draft horse and given birth to young could never thereafter give birth to purebred offspring of her own kind. Similarly, a pedigreed bitch having once mated with a mongrel and given birth to young would thereafter be unable to give birth to purebred offspring when mated with a dog of her own pure breed. It is understandable how such an erroneous idea came into existence, for it is nothing more than an *idea* which is erroneous, as simple attention to the facts themselves might have demonstrated. The idea of telegony, however, is a striking example of the truth that ideas based on erroneous theories will make men insensible to the facts that are before their eyes and cause them to see facts not as they are but as their belief says they should be. To repeat, it is understandable how such an erroneous idea came into being when it was believed that heredity was carried in and transmitted through the blood, for if the first sire’s hereditary qualities entered into the female’s blood stream, her subsequent offspring by subsequent sires would acquire some of the “heredity” of the first sire through its presence in their mother’s blood. Today, we know that this idea is entirely erroneous, on both theoretical and experimental grounds.

Breeding experiments on animals have shown that telegony is a nonexistent phenomenon. Nevertheless, in pre-genetics days it

was one way of explaining the fact that some offspring sometimes showed characteristics that were not present in their parents. This was especially true of many domestic animals in which traits would appear as a result of the fact that each of the parental animals carried the genes for them in recessive state while not exhibiting the traits themselves. But the simplest explanation was to attribute such traits expressed in the offspring to the effects upon heredity of some earlier sire.

In man the idea of telegony has reappeared in modern times principally in Germany. There in 1919, immediately after the First World War, an anti-Semitic novel was published by one Herr Artur Dinter, entitled *Die Sünde wider das Blut* (*The Sin Against the Blood*), in which the author argued by implication that the blood of the German people was being "poisoned" by the after-effects of admixture with Jews. This novel sold by hundreds of thousands, and there can be little doubt that it made Hitler's later promulgation of the same doctrine all the more easily acceptable by the German people.

Since many people still believe that the seed comes from all parts of the body and is carried in or is merely a specialized portion of the blood,* it should be stated once and for all that neither the male nor the female seed is ever carried in the blood or ever forms any part of the blood. The seed is carried in their germ cells, the egg of the female and the sperm of the male, in the one case from the ovary to the fallopian tube and in the other case from the testes through its ejaculatory ducts into the male organ. Blood is not in any way involved.

The erroneous belief in the inheritance of acquired characters, namely, the idea that changes in the structures or functions of one or both parent's bodies could be transmitted to their offspring, was based on this false notion concerning the relation of the seed to the blood. If the blood gathered the seed from every part of the body, then it was reasoned that any modification of the body would be reflected in the seed, and hence would be transmitted to the offspring. Just as the evidence is entirely contrary to this view of the relation of seed to blood, so the evidence is entirely contrary to the existence of such a phenomenon as the inheritance of acquired characters.

*We find this view clearly stated by the Father of Medicine, Hippocrates (c. 460-377 B.C.), in his book *Airs, Waters, and Places*, xiii, 14.

But enough, I hope, has been said about the myth of blood as the carrier of hereditary characters to exhibit it to the reader for what it is: an erroneous and harmful idea, perfectly understandable in its development, but belonging in the same class with the ideas that flies are generated from putrefying meat or that the touch of kings is capable of curing scrofula and other diseases. As a synonym for heredity the very word “blood” should be abandoned.

Heredity and Environment

HEREDITY or Environment: Which Is Stronger? This was a very popular subject for debate during the school days of almost everyone. Such debates were usually quite exciting, and since the discussants were not generally too well informed, confidence and passion would tend to conceal the insecure grasp of the facts and the words would fly. Nothing would be resolved except, possibly, the resolve to acquaint oneself with the facts. Not too many seem to have made such a resolution, for the debate still goes on in spite of the fact that the question, Heredity or Environment: Which Is Stronger? is a spurious one—spurious in the sense that it opposes and treats as separate two things that, in the context in which they are generally discussed, are not naturally either opposable or separable.

In practice we often do behave *as if* things that are not separable are capable of being separated, and in this way we are often able to discover how much of one is involved in the other. This is a common daily practice. But it is quite another thing to assume that two things we have arbitrarily decided to treat as separate are in fact separate in their action. Usually what we are dealing with, especially when concerned with living things, is not so much *action* as *interaction*, and certainly it is of the greatest importance to grasp the fact that living things are not results of actions but of *interactions*. Things acting by themselves do not in living organisms produce developmental changes; they do so only in interaction with other things. For example, there is no such thing as a gene that acts as such, by itself. Genes interact with other genes

and with the environments in which they occur. The environment interacts with the genes and the genes interact with one another. Thus we can recognize that the environment and genes exist separately as such, but with respect to the development of the organism they never exist separately but are always in process of interaction. Neither genes nor environment alone can produce development. Both genes and environment in interaction are necessary for development to take place. It is the *interaction* that is the important part of the process, for the genes and environment are not simply parallel processes. In the interactive process within the organism, the environment affects the action of the genes, and in turn the genes in interaction with one another and the environment produce changes in their own environment. This is a short statement of the genetic developmental process, the detailed expansion of which could easily occupy the space of a hundred books as large as this one. The point to grasp here is that development is an interactive process between the hereditary packages of chemicals, the genes, and the environment, that is to say, whatever is capable of interacting with the genes.

Development consists of changes in size, complexity, kind, number, position, shape, composition, and functions of the parts of the organism. Every one of these facets of development is under the combined influence of the genes and environment. The organism represents the end effect of the interaction between the two. (Remember that when we say "between the two" we are referring to a vastly complicated chemical series of processes in interaction with an equally complex series of environmental processes.) In development from the germ cell to the adult a tremendous number of quantitatively and qualitatively different interactions of elements must occur, and during the progress of these interactions almost anything can happen along the way to modify their expression. The fact that this is so often enables us to disentangle something of the nature of the factors, both genetic and environmental, that have entered into the formation of a trait or character at the other end of its developmental history. On all this we shall have most light thrown for us by studying the actual process of development in specific examples.

The idea that development simply represents a sort of unfolding of what is already preformed in the egg or sperm or the combination of both is one that was held in the seventeenth and

eighteenth centuries and has persisted down to the present day in the minds of many people in a more or less vague sort of way. This idea is known as the *preformation hypothesis*, and in its day was held by quite a number of respectable scientists. In the days when microscopes were very primitive, imagination had to make up for what the lens was unable to do, and so the sperm was seen as a homunculus or "little man" all ready and preformed in all its parts, simply waiting for the opportunity to unfold like the elements in the bulb of a flower. Several of these investigators actually showed a figure of a little man seated in the head of a sperm (see fig. 9).

The opposite of this theory, known as *epigenesis*, was developed by a twenty-six-year-old German, Kaspar Wolff, in 1759. In his book *A Theory of Generation* he suggests that organization of the embryo occurs absolutely spontaneously by means of an "essential force" acting on the undifferentiated germ, the embryo resulting from the coagulating power produced by the warmth and development of the blood vessels.

The mistake in the doctrine of preformation lay in supposing that the germ was a miniature organization of the adult; the error of epigenesis was in maintaining that the germ lacked any organization at all.

The "little man, what now?" theory, as the doctrine of preformation may be called, and the "spontaneous-combustion" theory, as we may describe the epigenetic theory, now belong to the realm of the history of science. No scientist subscribes to them today, but in one form or another these ideas linger on in the minds of some people. We can dismiss such ideas from curs.

What Is Heredity

It is extremely important to grasp the relationship between heredity and environment, and there is nothing in the least difficult about doing so. When we are able to perceive (1) what heredity is, (2) how development comes about and may be modified, and (3) what we human beings can do about both heredity and development, we can understand what the relationship between heredity and environment really is.

To the question, What is heredity? the answer usually returned by many authorities is that it is the innate endowment or equip-

ment of the individual. It is not what the individual is born with, but what he receives at fertilization, his genetic endowment or pattern of genes, the *genotype*. However, as soon as we begin considering the role that the genes play in the development of the individual as a functioning organism, structurally, physiologically, and psychologically, we perceive that the genes can do none of this, and there can be no development, without the interacting environment.

The end product that the organism represents at birth is the result of the interaction of the genes with each other and with the varying environments within the developing organism that they are in process of helping to form. In addition, there is the environment directly provided by the mother—we call this the *maternal* or *uterine environment*. This is of quite a complex nature and is only now coming to be recognized for its important role in the development of the organism. We shall return to this. Then there is the environment outside the mother, which is capable of affect-



FIG 9. Hartsoecker's drawing of a human spermatozoön showing a homunculus, or miniature man, 1694.

ing the developing organism within the womb. Radiation is an example. Loud sounds constitute another. These things are capable of reaching and affecting the development of the fetus directly without any intermediation on the part of the mother. And as we shall see in the next chapter, there is good evidence that many externally originating stimuli of various sorts are capable of seriously affecting the development of the organism.

The Dangers of Overvaluing Genes

It is a vast and confusing oversimplification to think of an individual's heredity as something limited to the genotype or the innate endowment. It is also dangerous and harmful to think in this way, for if it is believed that heredity is what is given in the individual's genes, then the conclusion is likely to be drawn that what the individual is going to be, or what he is, is wholly determined by the genes, and therefore there is nothing one can do about heredity. This is a common way of thinking about heredity, and it is quite false. Unfortunately, quite a number of "authorities" have fallen into this error. This error is known by various names: "the biologicistic fallacy," "the pathetic fallacy," "the nothing-but fallacy," and "the reductionist fallacy." "The biologicistic fallacy" describes the erroneous belief that man is nothing other than the product of his biological make-up. "The pathetic fallacy" is used here as an expression of sympathy with those who should know better for falling into so pathetic an error as the biologicistic trap; "the nothing-but fallacy" refers to the belief that man is nothing but an expression of his genetic or animal structure, and "the reductionist fallacy" the error of reducing man to his innate structure.

The danger of thinking of heredity as equivalent to genetic endowment lies in the fact that one is likely to think that this is all that heredity is. With such a conception of heredity the line of reasoning then follows that since the genes determine heredity, there is nothing much one can do about the heredity of the individual once he is conceived or after he is born. This is the kind of belief that makes it possible for a doctor or anyone else to say, "Since this is a hereditary condition, there is nothing that can be done about it." Another word that is often used in a similar way, instead of "hereditary condition," is "constitutional." If a condi-

tion is "constitutional" the frequent assumption is that there is little or nothing that can be done to modify it. And if one wants to be very learned one can call the condition a "constitutional idiopathy" and that *really* closes the door on doing anything about it.

It has been truly said that while high walls do not a prison make, scientific terms frequently do. Many an individual and his family have been needlessly caused to abandon all hope by the pronouncement of the verdict that a disorder was due to heredity. It is, of course, perfectly true that there are some disorders that are due to the action of genes about which we can do little or nothing at the present time, but this does not mean that this is true of all conditions that are caused this way. Nor does it mean that those about which we can do nothing at the present time must forever remain hopeless conditions. On the contrary, there is every reason to believe that when we have more fully come to understand the manner in which these conditions come about, we may be able to control their manifestations. In many instances, diabetes is a genetic disorder. When I was a youth, it was incurable, but since 1922, owing to the discovery and isolation of insulin by two Canadian investigators, Banting and Best, the sufferer of diabetes has obtained relief by being injected with the insulin in which his body is genetically deficient. Insulin does not cure or alter the defective heredity, but the body of the diabetic responds with normal carbohydrate metabolism to the environment which provides him with insulin. Hemophilia or "bleeder's disease," a hereditary condition affecting some males which takes the form of intractable bleeding from a slight wound, sometimes fatal, was until recently unrelievable. With the isolation of blood-clotting substances in recent years, it has for the first time become possible to help some of the sufferers from this unfortunate condition. We can already foresee that it will not be long before hemophilia becomes as relievable as diabetes. The treatment of pernicious anemia with liver is another case in point. Indeed, theoretically there is no chemical which genes are capable of producing that cannot be introduced into the body in some other way. Hence, inadequacies due to defective genes should be replaceable by chemicals originating outside the body. The point, of course, is to know what is the nature of the chemicals that are responsible for the defect.

There are many kinds of other hereditary conditions which may be similarly alleviated by environmental means. Tuberculosis, allergies, defects of vision and hearing—these are some of the common genetically conditioned disorders which have been greatly helped by control of the environment. In the case of hereditary defects of vision and hearing, the benefits conferred by eyeglasses and hearing aids can be at once appreciated.

At the same time attention must be drawn to the fact that there exist many genetic deficiencies which cannot be remedied in any environment. Furthermore, that every time we do discover a means of compensating for the genetic deficiency we thereby relieve the only control we have for *mutation pressure*, and thus increase the frequency of the gene, and therefore the number of individuals who are affected by it.

The danger, then, of making an exclusive identification of the genes with heredity is that it leads one to believe that innate endowment is equivalent to fate, and that the pattern of genes an individual inherits determines his destiny. Such a belief is a modern form of the older doctrine of predestination. There is little more truth in the one than there is in the other. When, then, we think of the action of genes in development it is desirable to remember that that action is an *interaction* with the environment, for development is a continual process of the adjustment of cells to their environment.

The Action of Genes

Development is not something static; it is a dynamic condition which varies with the conditions. What any cell shall become, what part of the body it will form, is not determined only by the genes it contains but depends on the conditions surrounding it, on its relation to other cells. This constitutes the answer to the question which arose on an earlier page, namely, how is it possible that cells which contain identical genes, the same materials, form the numerous different parts of the body?

Genes furnish the essential chemical substances necessary for development, and in interaction with other genes produce changes which further serve to constitute part of their environment. The cells, in their molecular structure, shape, stress, pressure, surface tension, the enzymes to which they give rise—that is, chemical

substances that speed up the reactions in other chemical substances—are in a continuous state of adjustment and adaptation. Though they interact, the genes in the nucleus remain the same, but the composition of the protoplasm outside the nucleus (the cytoplasm) is differentially elaborated. Hence, we see how very important the role of environment is in all heredity. The structure of the genes sets the specific limits of development; that is to say, their inherent composition is such that only an elephant can develop from elephant genes, and only a man can develop from the genes of human beings. The genes also set the maximal limits to which the various differentiated cellular masses can normally develop. Abnormal conditions can disturb each of these limits: In the case of specific characters, the organism develops in such a manner as scarcely to resemble the species which gave it birth; such organisms, generally known as “monsters,” rarely survive to birth, and when they do they are likely to die shortly thereafter. In the case of the growth of cells into tissues, all sorts of abnormalities of growth and development may occur affecting either parts of the organism or the organism as a whole. Oversized or undersized parts of the body, or decreases or increases in size of the body, are familiar examples.

In many cases of dwarfs and giants it can be clearly shown that it is not so much the genes concerned with the size of the individual that are at fault, but rather the environment in which these genes develop, an environment which is usually provided by certain secretions (*hormones*, or chemical messengers) from several of the ductless glands, notably the pituitary gland situated at the base of the brain. We shall deal with these cases more elaborately later in these pages. Here we are engaged in discussing the general principles involved. From the analysis of cases of monsters, dwarfs, and giants we perceive how important the environment is in regulating the manner in which the genes respond in the process of development. As we shall see, no characteristic of man (or of any other living thing) is exclusively the product of genes or exclusively the product of environment, but the product of the interaction of each with the other.

The individual, then, does not inherit characteristics. When we speak of “inherited” eye color or “inherited” nose shape, this merely represents a figurative way of speaking. Such characteristics are no more present in the germ cells than are the parts of an

airplane in the various materials out of which it is constructed. Those materials have to go through a vast number of complicated environmental processes before they can be put together to make an airplane. Exactly the same materials, with or without the addition of some others, can by other environmental process be turned into an automobile or a cabin cruiser. The same materials in different environments may result in different end products. And so it is with heredity. The same sets of genes in different environments may result in the different expression of characters. Heredity does not transmit characters. What heredity transmits are the responses that the genes have made in the developmental process to the environments in which they have interacted. In a sense, genes may be compared to tools and the environment as the materials upon which they act. The genes can only do as much as (1) their particular limits enable them to do, and (2) the opportunities provided by the material. We can turn this around and regard the environment as the tools which work upon the genes, the material. Poor tools won't do justice to good material or to poor. Good tools will do as well by the material as the material permits, whether it be good or poor.

We come to understand, then, that genes are most usefully looked upon as chemical packages of potentialities. To what extent those potentialities are realized will depend upon the environmental conditions under which they develop. For this reason whenever one thinks of heredity as a process it might be helpful to substitute the idea of potentialities for it, for this immediately suggests the idea of possible states that can only be brought into being through the stimulus of the environment.

The upshot of all this is of the greatest importance for us as human beings, for where we control the environment, we to some extent control heredity. Heredity, it has been said, determines what we can do, and environment what we do do. The limits of what we can do are determined by the genes, but it is the environment that determines the extent to which the potentialities within those limits are realized. We do not, therefore, stand helpless and impotent before the implacable fate which heredity is misconceived to be. On the other hand, through the intelligent management of the environment there is a great deal that we can do about it, as we shall see in the pages that follow.

To the question, therefore, which we set out to answer in this

chapter, What is heredity? we may best make the answer that definitions are not properly meaningful at the beginning of an inquiry, but only at the end of one, and it is therefore only at the end of this book that we shall be able to give a reasonably complete answer, but at this juncture we may sum up our discussion thus far by saying what we have found heredity to consist of. First and foremost, heredity consists of the innate endowment of genes acquired by every individual at conception. It is these genes alone that are transmitted, and nothing else is transmitted in the genetic process. This, strictly speaking, is what is meant by "heredity." But as we have already seen, genes do not work their effects in a vacuum. Indeed, they are unable to work at all in the absence of environmental stimulation and they are to varying extents dependent for their very action upon environmental conditions. These environments are: (1) the intercellular environments in which the genes interact with each other, (2) the uterine or environment within the womb, and (3) the extrauterine environment, that is, the environment of the external world outside the womb. Each of these environments is extremely complex, and we shall be discussing their nature throughout the remainder of the book.

One thing more: When, in this book, we sometimes speak of the "inheritance" of blue eyes or of this or that character or trait, or when we say that a character or trait is "inherited" as a dominant or a recessive, it should be remembered that these are only shorthand ways of speaking. Characters or traits are, of course, not inherited as such, but only the genes connected with their development. The relationship between heredity and characters or traits is not static, genes do not determine characters in a one-to-one, invariable manner. On the contrary, the relationship between heredity and traits or characters is a dynamic one, with genes influencing development through chains of physiological responses of the organism *to its environment*. The same genotype in different environments may give rise to quite different responses.

The Effects of Environment upon the Developing Human Being in the Womb

IT HAS BEEN KNOWN for many years that it is quite possible to change the form of various animals by altering the environment in which they develop. One of the early classical experiments illustrating this involved the larval form of the small boy's delight, the common Atlantic Coast minnow (*Fundulus heteroclitus*). It was shown that if a salt like magnesium chloride is added to the sea water in which the eggs of this fish develop, many of the embryos and young fish develop a single eye instead of two normal eyes. In some individuals the eye is in the middle of the head, and in others it develops on only one side.

In ordinary sea water minnows develop two eyes, but in sea water that is slightly higher in magnesium salts minnows, with exactly the same genetic constitution, develop only one eye. This does not mean that such minnows will thereafter transmit only one eye to their offspring; on the other hand, they will always transmit the genes for two eyes and their offspring will develop the normal two eyes when they undergo development in normal sea water. The minnows have inherited a genotype for a certain set of characteristics which will go to form the visual apparatus, but it is clear that the form in which that genotype will be expressed is to a large extent dependent upon the environment with which it interacts. Genes, as we have seen, will respond to the environment only in the manner which that environment enables them to do so. What a given set of genes will produce, therefore,

depends not upon themselves but upon their interaction with the environment. Under different environmental conditions the same genes will produce different results. It is therefore not true that because an individual has inherited a given set of genes, he is thereby destined to develop certain characters and no others. The kind of characters he will develop will depend upon the kind of environments in which the building blocks of those characters undergo development. In the present chapter we shall deal with the evidence concerning the relation of changes in the environment of the human fetus to its development.

For many years it was believed and taught that the human fetus is so well insulated within the womb from the rest of the world, including its mother, that what went on outside the womb was, on the whole, unlikely to affect the fetus. Anything capable of affecting the fetus, it was believed, had to be pretty considerable. In recent years we have learned that this is quite untrue, that, on the contrary, the human embryo—the term we give to the human organism from conception to the end of the eighth week—is extremely sensitive to all sorts of changes in the mother and in the external world, and that as it develops as a fetus—the term for the conceptus from the end of its embryonic period at the eighth week to the time at which the organism is born—it continues to be sensitive to innumerable environmental changes, though less so than the embryo. This follows a general law of embryological development, namely, the younger the developing organism the more likely it is to be seriously affected in development by disturbing conditions. There are periods in the development of the organism which are known as critical developmental periods, so called because during these periods fundamental developmental changes are occurring which depend on perfect timing and the correlation of many different processes. Any disturbing factor that is introduced into the developmental process at such a time is likely to produce structural and functional disorders in varying degrees and of various kinds. It should be understood that the “developmental process” is the name we give to the orderly procedure by means of which the normal structures and functions are being built up. These processes involve not only the constructive ones of building, but also certain limiting processes which perform the tasks of guiding, checking, and channeling the building-up processes. All these processes can be affected by changes in the en-

vironment in which they are going on. And this is true of man as of all other living things.

Since a great many physical and mental defects in children are believed to be due to disturbances occurring during the prenatal (before birth) development of the organism it is important to know how these come about, for with the knowledge thus gained we will be in a better position to control the situation.

The effect of environmental factors upon the development of the prenatal organism may be considered under the following ten headings: (1) maternal age, (2) maternal parity, (3) maternal dysfunction, (4) maternal sensitization, (5) nutritional effects, (6) infections, (7) drugs, (8) physical agents, (9) emotional factors, and (10) other environmental factors.

1. Maternal Age

Conception takes place in a female. That female has had a history. That history has extended over a number of years. A female's past history as a growing and developing organism is closely related to her reproductive capacity. The age at which a female becomes capable of conception is, on the average, about three years after the first menstruation (menarche), which puts the average age at which a female becomes capable of conception at about sixteen and a half years. This does not mean that many girls cannot become pregnant before that age. Many can and do, but most in fact cannot and do not. It is known that girls who conceive at an early age experience a larger number of miscarriages and stillbirths than those who conceive at more efficient ages. Not only do more of their babies die at birth, but the youngest mothers have the highest mortality rates, and a large percentage give birth to defective children. The younger mothers (in the age group generally under seventeen) are generally physiologically incompletely prepared for the process of reproduction, hence, the high frequency of unsatisfactory terminations to their pregnancies.

Studies of the reproductive development of the female show that from every point of view the best period during which the female may undertake the process of reproduction extends *on the average* from the age of twenty-one to about twenty-six years of age. During these years, on the average, the female is at her optimum period of development. All the hormones necessary for the

successful maintenance of the pregnancy are present at their optimum levels, and the organ systems are in their healthiest states. In mothers under seventeen years of age the hormones have not yet reached the optimum levels, nor have the reproductive organs—the organs serving the functions of bringing the pregnancy to a successful conclusion—reached their optimum growth and development. It is for these reasons that the younger mothers have a higher maternal and infant mortality rate than the mature mothers in the age group of twenty-one to twenty-eight years. The mothers between seventeen and twenty and their offspring do better than the mothers in the younger age group but not as well as the mothers and their offspring in the twenty-one to twenty-eight age group.*

Very gradually from the age of twenty-nine onward there is a rise in maternal and infant mortality rates; the stillbirth and miscarriage rates rise, and so do those for the number of defective children. From the age of thirty-five years onward there is a sudden jump in the number of defective children that are born, especially of the type known as *mongoloids*, so called because of an alleged superficial but quite unsoundly based resemblance to people belonging to the Mongoloid major group of mankind. At one time it was suggested that such defective children were “throwbacks” to a Mongoloid stage of development. This suggestion has no scientific merit or validity whatever.

Mongoloid children may or may not have the fold of skin over the inner angle of the eye (epicanthic fold) or the flat root of the nose that goes with this, but they do have smallish heads, fissured tongues, a transverse palmar crease, with extreme intellectual retardation. Their I.Q. ranges between 15 and 29 points, from idiocy to an upper limit of about seven years. Mongoloids are cheerful and very friendly personalities, with often remarkable capacities for imitation and memories for music and complex situations which far outrank their other abilities. The expectation of life at birth is about nine years.

Half the known cases of mongolism were born to mothers of thirty-five years or more, and 1 out of every 400 births or so occurring in the United States is a mongoloid. The causes of

*For a detailed discussion of the facts, see Montagu, *The Reproductive Development of the Female*.

mongolism are obscure, but most authorities agree that the physiological aging of the maternal organism, resulting in a progressive decline in the functions of her reproductive system, are almost certainly involved, and that this, among other things, takes the form of inadequacies in the secretions of the hormones of the ductless glands. Since these hormones are necessary for the proper development of her eggs, their failure to appear in sufficient quantity has a severe retardative effect upon the development of the fetus. An inadequate hormonal environment may occur in pregnant women at any age, in which event they are likely, in a high proportion of cases, to give birth to a mongoloid. The mere accumulation of years is not in itself a cause of mongolism or of any other disorder. There is promise that the knowledge yielded by the study of the endocrine status of women during pregnancy may enable us to reduce considerably the number of mongoloids born. Such studies, by enabling us to recognize that some mothers are providing the embryo with an inadequate hormonal environment, or by discovering the women who are likely to provide such an inadequate environment for the embryo even before they become pregnant, may lead to the remedy by providing such women with all the necessary hormones. It is believed that most of the damage done to the embryo who becomes a mongoloid is done within the first nine weeks of pregnancy.

In mongolism we have the tragic example of what may be an adequately sound genetic system being provided with an inadequate environment with resulting disordered development in the embryo. That defective genes are sometimes involved is a possibility. It is, however, generally agreed that in most cases defective genes are not primarily responsible, even though we have several records of mongoloid identical twins, and fraternal twins in which one child was a mongoloid and the other normal. In such cases it still remains a possibility that the defective uterine environment was at fault, and that in the case of the fraternal twins one child managed to obtain a sufficient quantity of hormones while the other failed to do so. (Apropos of twins, it is also a fact that two-egg twinning increases with maternal age; that is, the twins are derived from separate eggs and are therefore not identical but fraternal twins.) Mongolism in some cases may be due to a combination of a defective gene or genes with low penetrance and a defective environment. In an adequate uterine environment the gene would not express itself; in an inadequate one it may.

In April, 1959, a group of English investigators found that in six mongoloids they studied the chromosome count was 47 instead of the normal 46. The significance of the extra chromosome in the causation of mongolism is not at present clear, but the fact that an extra chromosome is present is in itself highly significant. The extra chromosome reflects a failure in the normal process of mitosis, and this may be due to a number of conditions, aging of the ova being but one of them.

Hydrocephalus, or "water on the brain," a condition in which the head becomes greatly enlarged owing to the abnormal increase of the fluid in the lateral ventricles of the brain, is another condition which is associated with late maternal age at pregnancy. No adequate studies have been made on the mothers of such children which indicate causative factors in human beings, but experiments made on healthy female rats show that when a mother was fed a diet low in folic acid more than 7 percent of her young were born with hydrocephalus, suggesting that similar nutritional factors may be responsible in man.

It is known that disturbances due to sensitization such as result from blood transfusion and other conditions become more marked with increasing age of the mother, and such sensitization may seriously affect the development of the fetus. The age of the father has not been found to be significant as a possible factor in the production of fetal malformation. In connection with this point Jalavisto has brought forward some evidence indicating that the offspring of older mothers do not, on the average, enjoy as many years of life as do the offspring of younger mothers.

2. *Maternal Parity*

It is now well established that parity or the number of previous pregnancies a woman has experienced is significantly related to the survival of the infant, as well as to the frequency of fetal malformations. There is evidence that first-born children, as well as those born at the end of a long series of pregnancies, are less viable (able to live) than those born in between, irrespective of maternal age. Fetal malformations are slightly more common in children of primiparae (mothers having their first pregnancy) than in children who are the result of a second or third pregnancy.

Average birth weight and linear dimensions increase with the order of birth, the first-born being least in both these respects. At

this point it is necessary to underscore the fact that the findings we shall be mentioning in this section are all of a statistical nature, and that there are plenty of first-born and last-born children, as well as children who were born well after their mother's thirty-fifth birthday, who are in every way perfectly healthy. Number of children or age of mother should never alone deter a woman from maternity. It is, however, always a good idea to see a competent doctor for a complete checkup before embarking upon such an important undertaking.

3. *Maternal Dysfunction*

The term "maternal dysfunction" describes noninfectious functional disorder or disease in the pregnant mother. Such dysfunctions in the mother may seriously affect the development of the fetus. Pregnant women suffering from hypertensive disorders (high blood pressure) show a very high rate both of fetal loss and of maternal mortality, as well as of other serious conditions. In one series, reported by Chesley and Annetto, of 301 pregnancies in 218 women suffering from essential hypertension (disease due to no known cause), the gross fetal loss in the first hypertensive pregnancy reached the staggeringly high figure of 38 percent, more fetuses dying the higher the maternal blood pressure. There were a total of 13 maternal deaths, or 4.3 percent, some 200 times higher than occurs in general obstetrical practice.

Prediabetes and diabetes are disorders which are also known to affect the fetus unfavorably. Prediabetes is the name given to the condition in which a latent disorder of carbohydrate metabolism is present without the subject being aware of it. By giving a woman a glucose tolerance test, which is a test for determining the amount of sugar circulating in her blood after she has ingested a measured amount of glucose, it is possible to determine whether or not she is prediabetic. If after two hours of fasting the test shows more than 120 milligrams of sugar per 100 cubic centimeters of blood, the condition is almost certainly prediabetic. Prediabetic women generally have babies whose weight exceeds nine pounds, another indication of this condition. A large baby in a woman not known to be suffering from diabetes is cause for the suspicion that unless preventive measures are taken she will probably develop full diabetes some time in the not too distant future.

Diabetes is a disorder of carbohydrate metabolism as a conse-

quence of which the sugar taken into the body is not burned by the body and converted into energy. The disorder is due to a deficiency of insulin. Insulin is normally secreted by the pancreas. It is known that in diabetic mothers the fetus grows very rapidly, often reaching the birth weight of the newborn long before it reaches term, and so may present considerable obstetrical difficulty. The mortality rates of fetuses and newborn babies of diabetic mothers are extremely high. For example, in one series, reported by Gaspar, of 49 deliveries in 45 pregnant diabetics, there were 19 stillbirths and 6 newborn deaths, a mortality rate of 51 percent! But in a comparable series reported by Stephens, Holcomb, and Page, the mortality rate was reduced to 8 percent by careful attention to the requirements of the diabetic mother, including preterm delivery of the child by Caesarean section. There is evidence that Caesarean section in such cases is an unnecessary practice.

In women with prediabetes in whom a latent disorder in carbohydrate metabolism may become accentuated during pregnancy, the adverse effects upon the fetus are the same as those associated with maternal diabetes. In mothers with diabetes the stillbirth and newborn death rates are increased for many years prior to the development of diabetes proper, and during the five years immediately preceding its onset the fetal loss may exceed 30 percent. Drs. Carrington, Reardon, and Shuman have shown, however, that by the early recognition and treatment of the prediabetic mother, the fetal loss may be reduced to zero with nothing but beneficial effects to the mother and her offspring. Of 111 cases, 46 were detected early enough in pregnancy to permit adequate treatment and in these there were no fetal losses. Among the rest there was much neonatal morbidity, with a 25.6 percent infant mortality rate among the 39 cases unrecognized before delivery.

Hyperthyroidism, excessive secretion of the thyroid hormone thyroxine from the thyroid gland, is another condition which adversely affects the development of the fetus. In a recent report by Battarino and Capodacqua on a series of 46 pregnant Italian women with hyperthyroidism, some 30 percent of the fetuses died. In 6 women, who had had 15 pregnancies and who had undergone operation for their hyperthyroidism, the mortality rate of the fetuses was 40 percent.

Undersecretion of the thyroid hormone in the pregnant mother is responsible in most cases for goiter and cretinism in the infant.

There is some evidence, too, that maternal toxemias (general poisoning of the blood) and bleedings during pregnancy may in some cases produce defects in the brain of the fetus; and that a number of the children will become behavior problems or be mentally retarded.

4. *Maternal Sensitization*

In those cases where the genes of mother and fetus differ for the substances borne on the surfaces of the red blood corpuscles, the mother may become sensitized and produce antibodies which unfavorably affect the development of the fetus. This usually results in causing fetal anemia at a relatively late fetal age. The Rh incompatibilities constitute a well-known example of this. As Landsteiner and Wiener discovered, when the blood of the rhesus monkey is injected into rabbits or guinea pigs, a special serum is obtained. The serum will "clump" the blood of about 85 percent of all white persons. The factor in the blood which makes it clump in response to the serum is the *Rh factor* ("Rh" deriving from the first two letters of "rhesus"). Persons who have this type of blood are said to be *Rh-positive*. Persons who do not are *Rh-negative*. The exact way in which the Rh blood types are inherited is somewhat complicated. Three principal Rh factors are known, also several contrasting Hr factors, and at least ten major genes are involved. These result in twenty-one combinations of genotypes. Several of the new genes recently discovered increase the number of genotypes still further.

Understanding how the Rh factor operates and how it is inherited is extremely important in biology and medicine. As we shall see, it has meant the saving of the lives of a great many babies and the virtual elimination of many family tragedies that not so long ago seemed an inseparable part of their destiny.

When a woman who is Rh-negative marries a man who is Rh-positive, the first-born child of such a marriage is usually normal. However, during the following pregnancies the fetus may be lost by stillbirth. Or it may be born in such an anemic or jaundiced state that it lives only a few hours or days after birth. The infant dies from a blood disorder called erythroblastosis (Greek *erythros*, red; *blastos*, germ; *osis*, increase). The name means that the baby's blood stream contains numerous primitive red cells (erythro-

blasts) produced in response to destruction of the baby's red cells by the maternal Rh antibodies. The baby's normal red blood corpuscles, the erythrocytes (Greek *erythros*, red; *cyton*, cell), are subjected to wholesale destruction. Another name for the same disorder is hemolytic disease of the newborn.

The disorder may result when the fetus has inherited an Rh-positive gene from its father, while the mother is Rh negative. The fetus produces Rh-positive substances called antigens in its blood. If fetal red corpuscles pass through the placenta into the mother's blood, the antigens stimulate the production of Rh antibodies.* These Rh antibodies in turn pass through the placenta into the blood stream of the fetus. There they combine with the fetus's Rh-positive red blood corpuscles which may then break down. The result of such destruction of the fetus's red blood cells is that it becomes anemic, and there is too little blood for its needs. The usually associated jaundice, which yellows the skin, is due to the infiltration of the skin by the breakdown products of the cells. In severe cases the skin may take on a greenish color.

Fortunately this disorder does not occur as often as the facts of heredity might lead one to expect. Erythroblastosis fetalis occurs in only about 1 out of every 200 pregnancies. Actually, about 1 in 12 pregnancies involves an Rh-negative mother carrying an Rh-positive fetus. Therefore, 1 out of every 12 pregnancies might be expected to yield an erythroblastic child instead of the 1 out of every 200 that is in fact born. This fortunate discrepancy may be caused by the fact that in many cases the antigens from the fetus do not pass through the placenta. In other cases the mothers may not respond to the foreign antigens from the fetus. However, if we omit first-born children, who are seldom affected, the number of children actually affected would rise sharply to 1 in 17 or 18 pregnancies.†

*An *antigen* is a substance which can cause the formation of another substance, called an antibody, with which it reacts specifically. An *antibody* is a protein, produced in the body in response to contact of the body with an antigen, having the specific capacity of neutralizing or reacting with the antigen. Antibodies may or may not exert a protective function in this way, that is, by neutralizing infectious agents or reacting with proteins foreign to the organism.

† Why the first-born child is seldom affected is not definitely known, but there is good reason to suppose that it is due to the fact that the Rh-negative mother has to be adequately sensitized by the Rh-positive antigens of her first-born before her own body can respond with the appropriate antibodies. This sometimes happens with her first pregnancy, but rarely.

While in most cases erythroblastosis is caused by Rh sensitization, maternal sensitization to other blood factors is occasionally responsible. In fact, if the mother belongs to group O and the fetus to group A or B, the baby may be affected, in which case it is said to have A-B-O hemolytic disease.

Erythroblastic babies may suffer serious damage of the liver and brain. The damage to the brain results from the deep jaundice, which is associated with the reddish bile pigment *bilirubin*, a substance very harmful to brain cells. Since the red blood corpuscles carry the oxygen which is circulated to the body on their surfaces, destruction of these corpuscles also serves to reduce the amount of oxygen available to the brain and other organs. We know that oxygen reduction in adults can have serious effects on the brain. There is no doubt that the oxygen reduction created by the destruction of red blood corpuscles in the fetus can be damaging and even cause death. If the fetus is deprived of an adequate amount of oxygen during any stage of its development, the brain may be permanently damaged. This may explain how mentally deficient children sometimes occur in families in which there is no previous record of mental deficiency.

The method of treating erythroblastic infants is to give them an exchange transfusion of Rh-negative blood. It is important to remove the baby's damaged red cells as well as to inject fresh red cells. In an exchange transfusion the baby's blood is removed from an artery at the wrist at the same time that an equal volume of Rh-negative blood is injected into a vein at the ankle. The reason for using Rh-negative blood rather than Rh-positive is because the baby already has maternal antibodies in its blood and so, if Rh-positive blood were transfused to the baby, it would be destroyed too rapidly by the antibodies circulating in the baby from the mother. The Rh-negative blood helps the baby over the difficult period during which the antibodies from its mother circulating in its blood become exhausted.

The Rh antibody does not occur naturally in the blood; therefore its presence in the blood must be due to immunization. This may be brought about by a previous transfusion in Rh-negative females or by previous pregnancies involving an Rh-positive child.

Understanding the meaning of the Rh factor is of great practical importance. Every woman planning marriage should consult her

physician in order to learn the Rh types both of herself and of her prospective husband. There are various ways in which the undesirable effects of clashing Rh factors may be averted if doctors know about them beforehand.

Thus, once more, we perceive how a knowledge of the manner in which genes interact with each other to produce their effects upon the developing organism helps us to control those effects, and to master them.

There is much evidence that the fetus is affected by various protein bodies that pass to it from the mother through the placenta, and that excessive indulgence in certain proteins by the mother during pregnancy may produce all sorts of allergic reactions in the infant through sensitization of the predisposed fetus *in utero*. Ratner has shown that many cases of infantile allergy are probably due to this cause. He quotes the case of a mother who took seven to eight eggs a day throughout her pregnancy, mostly in the form of egg-nogs. Her infant developed a severe eczema three months after birth. At fourteen months the child was brought to Dr. Ratner who was then able to bring about a rapid improvement by the "absolute elimination of egg-containing foods."

Ratner quotes a dozen such cases, all with the same outcome and all involving the excessive consumption of eggs in one form or another by the mother during pregnancy. In other cases nuts were involved, and in still others the excessive consumption of milk by the mother during pregnancy was the responsible factor.

5. Nutritional Effects

Possibly the most important factor in influencing the development of the fetus is the nutrition it receives from its mother. The foods consumed by the mother are reduced to molecules which are able to pass directly through the placenta into the fetal blood stream. But there are many other factors, in addition to maternal nutrition, which affect the nutrition of the fetus. Among them are the mother's occupation during pregnancy, her health, general hygiene, and the sanitary conditions of her environment. These conditions generally reflect the socioeconomic status of the mother.

Fetuses and infants of mothers of low socioeconomic status are

smaller and have a higher mortality rate than those of mothers of higher socioeconomic status. In itself small size is not necessarily a handicap, but in many cases it constitutes a symptom of basic organic deficiencies which are destined to play an important role in the later developmental history of the organism. By the end of the first postnatal month children who may otherwise appear to be normal will sometimes exhibit evidence of a deficient intra-uterine environment in the form of lines of condensed bone which show up in X-ray films as thick white lines in the ankle or tarsal bones. These lines, corresponding to the lines of retarded growth seen in the long bones of older children and adults, and caused by periods of prolonged illness, indicate that disturbances in nutrition, from whatever cause, arising during prenatal life are capable of inscribing their effects very substantially upon the structure of the developing organism.

Numerous experiments carried out in recent years on lower animals suggest that maternal nutrition in the early stages of fetal growth is a decisive factor in the production of certain physical abnormalities. It is not at present clear, in most cases, how the damage is done, but the evidence strongly indicates that it is mostly because of the lack of certain vitamins or proteins and also because of some toxic disturbance occasioned by the mother's state of malnutrition. It has been found that maternal diets deficient in vitamins B, C, and D were common in women with a high frequency of malformed fetuses. Fetal rickets as a consequence of the depletion of the mineral reserves of starved mothers is a well-known phenomenon. It is also known that maternal diet deficiency in vitamin D predisposes the child to early rickets.

It is generally agreed that when nutrition during pregnancy is inadequate the fetus suffers more than the mother. If the mother's diet is good during pregnancy, then the infant is usually in good condition at birth. A number of different investigators have demonstrated the substantial importance of an adequate maternal diet during pregnancy for the health of the infant and the adult it grows to be—if it survives to be an adult.

In a Canadian study, carried out by Ebbs and his group, on 120 pregnant women who were on a poor diet compared with 90 pregnant women of the same socioeconomic status whose diet had been made good, it was found that not one of the 120 women on a poor diet showed any recognizable evidences of any defi-

TABLE II

COMPARISON OF 120 PREGNANT WOMEN ON A POOR DIET WITH 90 PREGNANT WOMEN OF THE SAME SOCIOECONOMIC STATUS WHOSE DIET HAD BEEN MADE GOOD

		Diet	
		<i>Poor</i>	<i>Good</i>
Prenatal maternal record	<i>Poor-Bad</i>	36.0%	9.0%
Condition during labor	<i>Poor-Bad</i>	24.0%	3.0%
Duration of first stage of labor	<i>Primipara</i>	20.3 hours	11.1 hours
	<i>Multipara</i>	15.2 hours	9.5 hours
Convalescence	<i>Poor-Bad</i>	11.5%	3.5%
Record of babies during first two weeks	<i>Poor-Bad</i>	14.0%	0.0%

Illnesses of Babies

During First Six Months

Frequent colds	21.0%	4.7%
Bronchitis	4.2%	1.5%
Pneumonia	5.5%	1.5%
Rickets	5.5%	0.0%
Tetany	4.2%	0.0%
Dystrophy	7.0%	1.5%
Anemia	25.0%	9.4%
Deaths	3.0%	0.0%

Miscarriages and Infant Deaths

Miscarriages	7.0%	0.0%
Stillbirths	4.0%	0.0%
Deaths:		
Pneumonia	2.0%	0.0%
Prematurity	1.0%	0.0%
Prematures	9.0%	2.0%

SOURCE: Ebbs, J. H., and others, "The Influence of Improved Prenatal Nutrition upon the Infant."

ciency diseases. But what they did find was that in every way the mothers and their offspring who were on a good diet did better than the mothers and their offspring who were on a poor diet. These facts are clearly brought out in Table II.

The facts set out in Table II are striking. They show that a diet

which was inadequate, although adequate enough not to produce any recognizable deficiency symptoms in the mother, seriously interfered with her reproductive efficiency and affected the fetus much more than it did her.

These findings were abundantly confirmed by Burke and her co-workers at Harvard. In the Harvard study, carried out on 216 mothers and their infants during 1930-1941, it was found that every stillborn, every infant dying during the first few days after birth, the majority with malformations of various kinds, with one exception, all prematures, and all functionally immature infants were born to mothers who had had inadequate diets during pregnancy.

The effects of malnutrition upon the fetus and infant were very evident in babies born in Europe during World War II. Birth weight and length of infant decreased and premature births and stillbirths increased, as did severe rickets, severe anemias, and tuberculosis. A German study is of significance here. This study by Dr. D. Klebanow reports on 1,430 newborns who had been born, during the years 1946-1948, to Jewish women who had been in German concentration camps and suffered great hardships previous to their pregnancies. All these women conceived *after* their concentration-camp experiences were over, in most cases several years later. Four percent of the newborn of these women showed congenital defects of various kinds. There were 12 with birthmarks (nevi and hemangiomas), 12 were mongoloids, 8 had clubfeet, 5 had hydrocephalus, 4 had supernumerary fingers or toes, 4 were born without brains (anencephaly), and so on in proportions greatly exceeding the normal expectancy. Normally one would expect to find 2 or 3 mongoloids in such a number of births; in this series the number is increased 4 to 6 times! This report has been interpreted as indicating that the damage done to the organism by malnutrition over a prolonged period, even after that period of deprivation is ended, may affect the maternal organism seriously enough to make the uterine environment she provides in her subsequent pregnancies in one way or another inadequate. However, the age of the mothers at conception may have been the significant factor in a number, if not in all, of these cases.

In those countries during World War II in which the food intake was rigidly and scientifically controlled, as in England, the health of children actually improved. Unfortunately, in other

TABLE III
NUTRITION OF MOTHERS DURING PREGNANCY AND BIRTH WEIGHT OF
THEIR BABIES

Nutrition of Mothers	Number	Average Birth Weight of Babies in Grams
Well nourished	63	3,098.3
Fairly well nourished	272	2,991.9
Undernourished	29	2,897.7

SOURCE: Acosta-Sison, H., "Relation Between the State of Nutrition of the Mother and the Birth Weight of the Fetus."

countries food shortages were not so ably handled. The fact that well-nourished mothers tend to have well-nourished babies, and poorly nourished mothers tend to have poorly nourished babies, indicates that the well-being of the child before and after birth is significantly influenced by the nutrition of the mother both before and at the time of conception, and also throughout the duration of pregnancy.

A recent extensive study by Harrell, Woodyard, and Gates has shown that the three-year-old children of mothers who had received a polynutrient vitamin supplement to their diets during pregnancy had an I.Q. that was 5 points higher than the children of mothers of the same socioeconomic group who had not received such a supplement. The I.Q. was 103.4 for the first group and 98.4 for the children of the nonsupplemented group.

In another study by Acosta-Sison it was found that mothers who had been well nourished during pregnancy had babies whose birth weight was more than 100 grams* greater than the babies of fairly well nourished mothers and more than 200 grams greater than the birth weight of babies of undernourished mothers (see Table III).

It has been shown by Burke and her co-workers that babies born of mothers who consumed 85 grams of protein daily during pregnancy averaged, at birth, as much as 3 pounds more in weight and were $2\frac{3}{4}$ inches taller than infants whose mothers consumed 45 grams or less of protein daily during pregnancy.

* A gram is about $\frac{1}{28}$ of an ounce.

Normally ingested food proteins are broken down into amino acids before they are absorbed into the circulation, but it is now known that unsplit proteins may enter the blood stream directly from the gastrointestinal tract. Such antigens entering the fetus, and those antibodies which it receives by filtration through the placenta, will attach themselves to various tissue cells. When similar antigens are introduced into the baby's body after birth, an allergic reaction may ensue, resulting from interaction of the protein and fixed tissue antibodies. The fixation of the antibodies may be to the skin, the gastrointestinal tract, the nervous system, the respiratory tract, and so on, and it is through one or other of these systems that the sensitized individual will react. Such allergic reactions may persist for many years.

The evidence is now fairly substantial that mothers who have an excessive craving for particular foods during pregnancy may by their ingestion produce various allergic sensitivities which show up in their children after birth.

But enough has been said on nutrition. Food is what helps cells to grow and develop, and cells are what help organisms to achieve growth and development. Hence, the fundamental importance of nutrition in development.

6. *Infections*

The more we investigate these matters the more we find that both viruses and bacteria are capable of passing from the mother to the fetus and of interfering with the development of the child and doing considerable damage to the latter. At the same time we also know that when such viruses are specially prepared for immunological purposes and are injected into the mother the maternal antibodies will enter the fetus and confer passive immunity upon it for at least several months after its birth. We have only recently learned this, for example, in connection with the widespread immunization of the population against poliomyelitis. However, similar knowledge in connection with measles was already in existence as early as 1702, and Edward Jenner, who introduced vaccination into England at the end of the eighteenth century, was aware of the fact that immunization of the mother against smallpox seemed to confer passive immunity against the disease upon her infant. This soon wears off and the baby must then be vaccinated in order to become actively immune.

Infections with the organisms producing smallpox, chicken pox, measles, mumps, scarlet fever, erysipelas, and recurrent fever have long been known to be transmissible from mother to fetus. There is also good evidence that the virus of influenza A can produce serious deformities in the developing embryo. German measles (rubella) is an example of a virus disease which, contracted by the mother during the first twelve weeks of pregnancy, is capable of producing gross developmental defects as well as death in the embryo or fetus; a high proportion of surviving infants suffer from such conditions as cataract and deafness with mental defect. Between 12 and 20 percent of fetuses will be adversely affected. If the mother contracts German measles during the first twelve weeks of pregnancy she has about an 8-to-1 chance of giving birth to a developmentally defective child.

Since the organ-forming period of the human embryo extends from about the fourth week to the end of the tenth week this is the period during which invading organisms may be expected to do most damage. Since the virus of German measles is capable of doing so much damage during this period, it has been reasonably suggested that it would be a good idea to have girls deliberately infected with the virus, and thus immunized, before they reach childbearing age.

There is evidence that poliomyelitis contracted by the mother during the first three months of pregnancy may have serious effects upon the development of the fetus. The evidence is unclear for the ill effects of measles, though the fetus may be born with a clear case of measles including the typical skin rash and other symptoms.

The bacterium causing syphilis (*Treponema pallidum*) can actually enter the embryo. If this happens miscarriage occurs. If the bacterium enters at a later fetal age, the child is born with signs of congenital syphilis—blindness, deafness, defects of the heart—or the disease may not show itself till later in the form of severe disorder of the nervous system.

Tuberculosis is also transmissible to the fetus from the mother by means of the *Mycobacterium tuberculosis*. The fetal death rate from tuberculosis is high, and those infants who are born with the disease usually die within the first year.

Malarial parasites are known to be transmissible from mother to fetus. Protozoan parasites, such as *Toxoplasma*, can be transmitted from mother to fetus and produce severe disorders of the

nervous system and eyes: meningoencephalomyelitis (inflammation of the brain and spinal cord and their membranes), microphthalmos (reduction in the size of the eyes), chorioretinitis (inflammation of the retinal and choroid layers of the eyes), microcephaly (reduction in the size of the head), and hydrocephalus, convulsions, and idiocy or mental retardation.* This disease, toxoplasmosis, occurs in adults in such a modified form that it is rarely recognized.

Various forms of cancer (malignant melanoma, choriocarcinoma) have been transmitted by the mother to her fetus.

Since congenital malformations constitute the second highest leading cause of death of newborn babies, and it is estimated that about 23 percent of the human race dies before birth or shortly thereafter from developmental defects, it will be appreciated how necessary it is for us to become aware of the importance of providing an adequate maternal environment for the developing human being.

7. *Drugs*

Drugs taken by the pregnant mother may seriously affect the fetus. Many cases of congenital deafness have been traced to the mother's use of quinine for malaria during pregnancy. Morphine has been reported in the infants of mothers who were morphine addicts. Inhalation of amyl nitrite by the mother for a few seconds has induced an increase in the fetal heartbeat, beginning during the third minute following the mother's inhalation. Subsequent inhalations produced a diphasic (excitor-depressor) response.

The recent obstetrical practice of dosing the pregnant mother with barbiturates and similar drugs prior to delivery may so overload the fetal blood stream as to produce asphyxiation in the fetus at birth, with either permanent brain damage or subtle damage that could lead to mental impairment. Fortunately, the trend today is away from heavy sedation.

It is known that a barbiturate derivative such as sodium secenal, usually prescribed as a sedative, when given to the pregnant mother will pass into the blood stream of the fetus and cause

*For further material on these disorders see Appendix A.

a depression in the waves measuring the electrical activity of the cells in the gray matter of its brain, and that this depression persists for some time after birth.

The effects of maternal cigarette-smoking upon the fetus may be considered under the present heading. The smoke, which does not have to pass into the lungs to get into the blood stream, is mostly absorbed directly into the blood stream of the mother through the mucous membranes of her mouth, nose, and throat. Tobacco smoke contains such noxious substances as nicotine, arsenic, carbon monoxide, furfural, pyridine, collidine, hydrocyanic acid, methyl alcohol, carbonic oxide, alkali, and various tar products. It is known that the nicotine absorbed by the smoker acts upon the aggregations of nerve cells (ganglia) of the autonomic nervous system and upon the respiratory center, that it increases the acidity of the gastric tract, produces acidity of the whole gastrointestinal tract, constricts the arteries, increases blood pressure, causes the heart to pump harder—to beat more rapidly—and produces irritation and congestion of all organ membranes.

Is there any evidence that the pregnant mother's smoking affects the fetus? There is. It has been found by Sontag and his associates that the smoking of one cigarette generally produces an increase, and sometimes a decrease, in the heart rate of the fetus. At seven months' fetal age, for example, when the fetal heart beats at the rate of 140 per minute, a few puffs by the mother on a cigarette is sufficient in many cases to send the heartbeat up by 40 additional beats, that is to 180 beats a minute, and sometimes to decrease the heartbeat by 17 beats per minute. The maximum effect is observed between the eighth and twelfth minute after the cigarette, and the response of the heart and blood vessels is more marked after the eighth month of pregnancy. It is quite possible that the products of tobacco entering the embryonic and fetal circulation adversely affect not only the heart and circulatory system, but also many other organs. The increase in cardiac and circulatory disorders in recent years may not be unconnected, in part at least, with the smoking of pregnant mothers.

In a recent study by Simpson of 7,499 mothers whose smoking habits during pregnancy were examined in relation to the frequency with which premature babies were born to them, some interesting findings were made. In this study a premature baby

was defined as one weighing $5\frac{1}{2}$ pounds or less. It was found that there was a significant correlation between the number of cigarettes smoked each day during pregnancy by these mothers and the frequency with which they were delivered of premature babies. Heavy smokers (more than ten cigarettes a day) had the highest prematurity rate; light smokers (between one and ten cigarettes a day) had a lower rate; the nonsmokers had the lowest prematurity rates. The prematurity rate, in fact, was approximately twice as high for smokers as it was for nonsmokers, being 11.4 percent for the smokers and 6.4 percent for the nonsmokers (see fig. 10).

8. *Physical Agents*

Differences in pressure *in utero*, whether induced through internal or external forces, differences in position, umbilical cord entanglements, and similar factors may more or less adversely affect the development of the organism. Deformity may be caused by faulty position, mechanical shaking, temperature changes; asymmetry of the head may be produced by pressure of the head downward upon the thorax. Wryneck (torticollis) has also been observed, and pressure atrophy of the skin indicates the kind of continuous stimulation to which the fetus may be exposed.

Exposure to massive doses of X rays within the first two months of pregnancy will in many cases produce abortion of the embryo. Where abortion does not follow, serious injury has been found to result in a percentage of cases. In a series reported by Professor Douglas Murphy, 75 children whose mothers' pelvic organs had received therapeutic doses of X rays during pregnancy were studied. Only 37 of these children were normal; 38 were malformed. There were 18 microcephalic idiots, 1 case of hydrocephalus; 4 suffered from other serious defects of the nervous system; 6 were premature, weak, and diseased; 2 were doubtfully mentally defective; 2 were stillbirths; and 5 were otherwise deformed. In other words 50.7 percent of these children were abnormal.

In April, 1959, a report was released by the New York State Department of Health which indicated that high natural radiation areas in that state were associated with a high congenital malformation rate of more than 20 per 1,000 live births for the

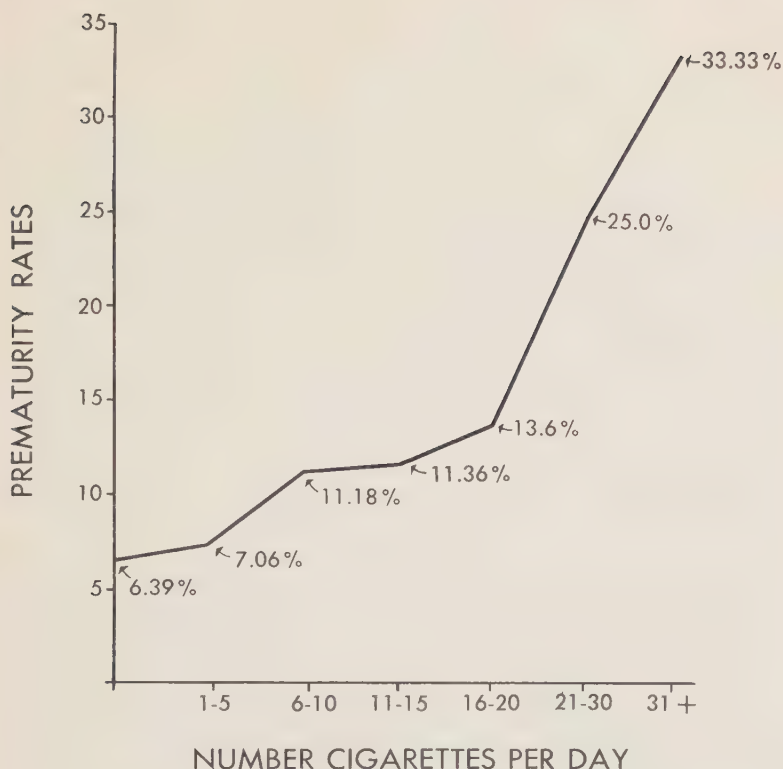


FIG. 10. Relationship of Number of Cigarettes Smoked a Day to Prematurity Rates. (After Simpson.)

period 1948 through 1955. The state-wide average is 13.2 malformations per 1,000 births.

In recent years we have learned that the fetus may respond in a convulsive or startled manner to sudden loud noises or vibrations, and that sound reaching it in any form may elicit increase in its activity.

It has, of course, long been known that the birth process itself may seriously affect the fetus, and that at this time damage to the brain or other organs may be produced by a difficult labor and attempts to deliver the child with instruments.

9. *Emotional Factors*

There is some evidence that emotional disturbances in the mother may affect both the structural and psychological development of the fetus. Emotional changes in the mother insofar as they produce chemical changes within the body of the mother may by this means cause the passage of excessive quantities of certain chemical substances into the developing embryo that produce disturbances of growth and development. Some evidence has recently been turned up suggesting that emotional disturbances during the first ten weeks of pregnancy may be responsible for the production of a large number of children with cleft palate. The bones which enter into the formation of the palate develop between the seventh and tenth week of embryonic age, and any disturbance at this time might well affect the development of these bones adversely. The means by which this might be accomplished is as follows: Maternal stress is known to cause hyperactivity of the cortex, or outer layer, of her adrenal glands (situated on the upper poles of the kidneys). Hyperactivity of the adrenals causes the secretion of the hormone hydrocortisone. Hydrocortisone can pass through the placenta into the fetus. When hydrocortisone is injected into mice or rats at the time when their palates are being formed, almost 90 percent of them are later born with cleft palates. The probabilities are high that a similar mechanism is at work in man.

Children born of mothers who have been seriously disturbed emotionally at some time during their pregnancy frequently exhibit psychological and psychosomatic irritability after birth. There is much evidence which strongly suggests that a human being's nervous system may even be permanently sensitized by the action upon it of excessive quantities of chemicals released as a consequence of the pregnant mother's emotional disturbances.

Emotional factors are known to play an important role in habitual miscarriage and sterility.

10. *Other Environmental Factors*

The direct and indirect effects upon the fetus of radiation from the fallout of atomic bombs is a matter important enough to de-

serve a separate chapter. The subject is therefore dealt with in Chapter 18. Here we may mention an environmental factor affecting the fetus *in utero* which may malform the human being for the rest of his life—iodine deficiency leading to endemic cretinism.

Endemic cretinism is due to a congenital deficiency of the thyroid gland which causes it to produce an inadequate amount of its normal secretion, thyroxine, which is high in iodine salts. In areas of the world in which the soil is deficient in iodine all food-stuffs derived from that soil are likely to be poor in iodine, thereby causing iodine deficiency in the individuals living in such areas (see fig. 11). Iodine deficiency will almost always result in a goiter. Mothers suffering from iodine deficiency are highly likely to give birth to children who are cretins. Cretins suffer from an almost complete lack of the thyroid hormone and are stunted both physically and mentally, are often more or less deaf, and in addition to



FIG. 11. Seven Cretins from the Urnatsch Almshouse, Appenzell Canton, Switzerland. The tall man is normal, the height of the woman immediately in front of him is 39 inches. (Courtesy of Dr. J. F. McClendon.)

a characteristic slouching gait, are frequently affected by other abnormalities. All the children of a goitrous woman need not, however, be cretinous, and if the mother moves to an environment where her children are brought up on foods having an adequate iodine content, they need never become cretinous. If she stays where she is, her children need not be cretinous if she is treated throughout pregnancy with iodine. Healthy persons moving into an iodine deficient area may develop goiter.

Cretinism is not always due to iodine deficiency in the mother; in some cases it is caused by a deficient gene.

Only a small fraction of the evidence has been cited bearing on the manner in which the development of the human being may be affected while in the womb, but this should be more than sufficient to show the importance of environmental factors in the development of every human being. The evidence we have considered in this chapter betokens something of a record of tragedy and unhappiness, but as a consequence of our increased understanding of the causes of such unhappiness, it becomes possible for us to conclude this chapter on a happy note. We know that heredity or constitution is not a mystery tantamount to fate or predestination, that on the other hand there is much that can be done to make the gestation period, the $266\frac{1}{2}$ days of pregnancy of the mother, a thoroughly successful one in the vast majority of cases. The health of our children is in our own hands. We can do something about it. If we eat sour grapes, our teeth may be set on edge, but there is no reason why our children's teeth should. We need no longer be doubtful or anxious about whether or not we are doing the right thing as parents. Abundant knowledge of the right thing to do is now reasonably available. All we need to do is to acquire that knowledge and to act upon it. The health of the infant and child begins at conception. This being so, care for the quality of the genes that enter into that conception, by the intelligent selection of mates, should be at least as important a consideration as care of its prenatal and postnatal environment. Once the conception has been brought about the care of the infant and child begins with caring for the human being developing in the womb. In this respect nothing is more important than the health and well-being of the mother who nourishes it.

Environment After Birth

WHEN A BABY is born he enters what is for him an entirely new environment, the postnatal environment, the environment of the world outside the womb. Having lived an entirely “aquatic” life in the womb, rocked, as it were, in the cradle of the deep of his mother’s amniotic fluid and living off the oxygen, among other things, transmitted to him across the placenta from his mother, he now has to adjust to breathing in atmospheric air, and instead of his food being supplied him in his blood, he now has to take it in through his digestive system. In a relatively short period of time—the period of birth—he is called upon to make literally scores of new adjustments. But however well he automatically makes these adjustments, if the environment fails him the newborn will not do well. The most important of all the elements in the human infant’s environment is other human beings, at least one. An infant must have oxygen, but this it normally obtains without any more effort than is involved in breathing. It must rest, sleep, and eliminate. All these things it is able to do without effort. It must also take in food and liquid. These things it cannot do without the provision of the necessary food and liquid by another human being. Usually it is the biological mother who provides the infant with its food and attends to its other needs. From the moment of its birth the quality of the mothering the infant receives, whether from its own mother or from someone else, is fundamentally related to the quality of its development physically and psychologically.

The Importance of Mothering

If the infant has enjoyed optimum conditions for development in the womb, his postnatal development may nevertheless follow a seriously disordered course as a consequence of inadequate mothering. Furthermore, we now have ample evidence indicating that as a result of inadequate mothering, some children will fail to grow and develop, and will die, especially during their first and second years. Such children present a pathetic picture of abandonment and hopelessness. They exhibit all sorts of regressive changes: pallor and wrinkling of the skin, dullness of the eye; they will lie motionless and quiet for hours or else cry for hours; they will regurgitate their food, suffer from diarrhea; they will virtually cease to grow and suffer a marked mental deterioration. Dr. René Spitz, in a classic study, reported his investigation of 239 children who had been institutionalized for one year or more, one group of whom were nursed by their mothers in an institution which he called the "Nursery." In the second institution, "Foundlinghome," the children were raised from the third month by overworked personnel, one nurse caring for from eight to twelve children. "Nursery" did not lose a single child through death, whereas in the "Foundlinghome" 37 percent of the children died during the two-year observation period.

"While the children in 'Nursery,' " writes Spitz, "developed into normal healthy toddlers, a two-year observation of 'Foundlinghome' showed that the emotionally starved children never learned to speak, to walk, to feed themselves. With one or two exceptions in a total of 91 children, those who survived were human wrecks who behaved either in the manner of agitated or of apathetic idiots."

The World Health Organization report entitled *Maternal Care and Mental Health*, written by Dr. John Bowlby and published in 1951, discusses all the material on this relationship between mothering and development up to the year 1951, and abundantly confirms the importance of good mothering for healthy development. It is now clear that a child who has not been adequately loved for any durable period during its first half-dozen years of life may suffer more or less severely not only during those first six years, but for the rest of his life.

Just as there are critical developmental periods in the organ-forming stage of the embryo's development so there are critical developmental periods in the personalization stage of development. Personalization is the process of personality organization, the way in which the individual comes to be organized as a behaving organism in relation to others and to himself. There are at least three or four such critical developmental periods, which may be described as follows:

1. The period during which the infant is in process of establishing an explicit co-operative relationship with a clearly defined person—the mother. This is normally achieved by five or six months.
2. The period during which the child needs the mother as an ever-present support and companion. This normally continues to about the end of the third year.
3. The period during which the child is in process of learning to maintain a relationship with its mother during her absence. During the fourth and fifth years, under favorable conditions, such a relationship can be maintained for a few days or even weeks.

Inadequacies in the mothering experience during these periods are likely to have different damaging effects upon the development of the child. These relationships are under intensive study at the present time, and it will be some years before we shall be able to correlate the behavioral changes with the critical developmental periods. They have been studied carefully in dogs, and it has been shown in them quite clearly that there does exist a significant relationship between critical developmental periods for social behavior and susceptibility to defective behavior in response to certain kinds of environmental stimuli. For example, it has been found that when puppies are weaned too early (at about three weeks), they tend to show the canine equivalent of thumb-sucking; they will suck either tails or ears or toes. When puppies are allowed a week or two more of nursing, they do not show such behavior. If one allows a puppy to be raised by its mother without the experience of human beings, say for the first twelve or fourteen weeks of its life, it then becomes extremely difficult to get the animal to relate to human beings.

If graylag goslings are exposed as soon as they hatch from the egg to a human being instead of to the mother goose, they will thereafter attach themselves to the human being as if he were the mother. They will form a similar irreversible attachment to any moving object. If one takes a newborn guinea pig away from its

mother before it has nursed and keeps it separated from the mother for some three days, it is found that the guinea pig cannot learn to nurse when it is returned to its mother.

These examples are but a few of the evidences for the existence of critical developmental periods in lower animals. These critical periods are undoubtedly genetically based, and from them we have learned not only that certain environmental stimuli must be received within a circumscribed period of time if normal development is to occur, but that if other forms of stimuli are substituted, permanently abnormal forms of behavior will follow. Insofar as it has been possible to do so, these conclusions have in every way been confirmed in human beings, often in the most dramatic and spectacular manner.

No one, for example, would have suspected that lack of a warm emotional environment could possibly interfere with the bone growth of children. But it does. And not only the bones, but every aspect of physical growth may be affected. These findings were made on a group of children in a Cleveland institution for dependent and neglected children by Drs. Ralph Fried and M. F. Mayer. They summarize their findings in the following words: "Socio-emotional adjustment plays not merely an important but a crucial role among all the factors that determine individual health and well-being . . . it has become clear that socio-emotional disturbance tends to affect physical growth adversely, and that growth failure so caused is much more frequent and more extensive than [is] generally recognized." These investigators found that there was only one treatment that could successfully overcome the retardation of growth suffered by the institution children they investigated, and that was removal to a warm loving environment. A similar finding was made by the Medical Director of Schools at Saskatoon, Canada, Dr. Griffith Binning. Dr. Binning, in a remarkable study on the effects of emotional tensions on the development and growth of 800 Saskatoon children, found "that events in the child's life that caused separation from one or both parents—death, divorce, enlistment of a parent—and a mental environment which gave the child a feeling that normal love and affection were lacking did far more damage to growth than did disease," that such an environment, indeed, "was more serious than all other factors combined."

The same children with the same genes in happier environments

would respond to those environments very much more happily than they were able to do in the environments in which they found themselves.

What is the heredity of such children? Clearly, children inherit postnatal environments as well as genes and prenatal environments, and the interactive consequences of these last two with the postnatal environments they inherit can be just as lethal and retardative, or stimulating and encouraging to the realization of the organism's potentialities, as the prenatal environment. It is extremely important to emphasize this point, for it is widely believed that heredity is "what you are born with," and that "what you are born with" pretty much determines what you are going to become. The two beliefs taken together are both too limited and too comprehensive. Heredity is what you are born with, in the sense that you are born with palm prints and sole prints, but it is also true that heredity includes what you are born *into*. The potentialities of each individual are dependent on genetic endowment, but these potentialities have a wider range than is generally observed, so that the environment *which is part of an individual's inheritance is also the means by which the heredity of an individual can be changed, modified, enlarged, contracted, and so on.*

The child's potentialities at birth have by far the greater part of their development to realize in the years *after* birth, and how those potentialities will be realized will again depend upon the nature of those potentialities in interaction with the environment to which they are exposed. It may not be possible to make a silk purse out of a sow's ear, but it is most certainly possible to depress the environment to such an extent as to make sow's ears out of materials that would otherwise have become silk purses. But that is a metaphor, the first part of it too frequently used by those who believe that genes determine the fate of men. The facts, however, enable us to make a more correct generalization: the genes or potentialities being what they are at birth, what a human being subsequently becomes is then, within the limits set by the genes, largely determined by the experiences to which he is exposed. Let us consider the facts.

We are all born with potentialities for speech, but whether we will speak at all is dependent entirely upon the kind of exposure we receive to speaking human beings. As children, if we only hear a word now and again and are not involved in the necessity of

learning the meaning of things, the probabilities are high that we will never speak. Some years ago in Ohio a little girl was found locked up in a closet where she had been kept for five years with her deaf-mute mother. At the age of five years when she was discovered or liberated, this little girl could not utter a word, but after some six months of intensive work with a speech expert she eventually learned to speak perfectly. She had, incidentally, received a great deal of love from her deaf-mute mother during their incarceration. None of us would ever speak unless we heard other people speaking. We possess the general potentialities for speech, but what language we will speak will depend entirely upon the language we hear spoken. And so it is with all our behavioral potentialities. How we will behave about them depends largely upon the manner in which we have been taught by the environment around us. Our very senses have to be taught in this way. Our tactile sense, how we see, hear—all these we have to learn.

Compared to nonliterate people we are very "thick-skinned." They often laugh at the length of time it takes a white man to discover that some insect has alighted upon his body, he generally being unaware of it until after it has bitten or stung him. They can usually feel an insect the moment it has alighted. Their sense of visual discrimination is markedly more highly developed than ours, simply because in their way of life it is vastly more important than in ours. But all human beings have to learn to see three-dimensionally and to evaluate the conditions of perspective.

The apparent simplicity of seeing is the result of a long learning process in which we eventually bring the whole of our past experience to bear on *how* and therefore *what* we see. It is possible to demonstrate this in a number of ways, but perhaps in none more convincingly than in the "distorted room" experiments of Ames and Cantril. In this room, the floor, ceiling, and back wall are all slanting. When one looks into this room with one eye through a hole one sees a normal rectangular room—that is, the *assumption* is made that it is a normal rectangular room whereas there are actually no right angles in the room. When we enter the room and move about in it, we understand that the eye did not in fact see what was to be seen. We prejudiced what we saw with the assumptions based on our former experience. Hence, we understand that our ability to see things is not entirely a matter of having healthy eyes, but depends, too, on the conditioning of our past experience.

All seeing is interpretation—and we interpret what we see according to the history of our experience.

How human beings will see, hear, and talk, provided they have the normal potentialities for these abilities, is, then, largely a matter of the experiences they undergo in the process of learning. With the exception of identical twins no two human beings are alike in their potentialities, and within the range of the normal the variation in the quality of potentialities is enormous among human beings regardless of ethnic affiliation, class, or caste. Thus, we can be quite certain that because of the great variation in genotypes even if we could make the environment identical for everyone, and no matter how favorable we made that environment, there would still be an enormous difference in the range of abilities exhibited. In fact, the variation in those abilities would be even greater than it is anywhere on earth today, for the more we maximize the opportunities for potentialities to express themselves, the more we increase the chances for the individual differences to express themselves. In a society in which no one can write, all its members are alike in their incapacity to write. Teach them to write and very soon marked differences will be discovered in the abilities of different individuals to write. By maximizing opportunities we do not reduce inequality in ability, we maximize it. This refers to physical as well as to mental development. It should be clear that by maximizing opportunities we increase the possibilities not only for increasing likenesses but also for increasing the differences that exist between individuals.

Physical Development

One of the best measures of physical development is increase in size. It has been known for many years that an unfavorable environment is capable of retarding growth. The kind of environment most generally involved is the socioeconomic environment, that is, the social environment and the kind of conditions that different amounts of money make possible. As might be expected, impoverished or low socioeconomic environments are associated with impoverished growth, and favorable socioeconomic environments are associated with favorable growth, and again this applies to mental growth as well as physical.

It has been shown that the rate of growth of children with two

unemployed parents is slower than that of children with only one unemployed parent, and that the children of employed parents have the higher rate of growth. Children in private schools on the average have a higher rate of growth than those in the public schools. The upper socioeconomic classes have less weight for height than the lower ones. Japanese who migrate as children to California grow generally larger than those who stay at home. Growth rates of children of middle-class homes are significantly higher than those of institutional children. Rate of growth is higher for rural than for urban infants in the first eighteen months of life. Efficiency of the mother, as measured by the tidiness of the home and children, has been shown to be significantly related to the growth of infants; the more efficient the mother the better was the growth of their children.

Sickness and mortality rates are highly correlated with socioeconomic status. These rates are similar during the first week of life, becoming steadily dissimilar during the first and second months in favor of the higher socioeconomic classes, so that the rates for the infants of the poorest parents become 3 to 4 times greater. By the end of the year the mortality rates of the infants of the poorest families are 8 to 10 times greater than the infants of middle-class families.

The higher morbidity (sickness) and mortality rates in infants of the poorer classes as compared with those affecting the more comfortably situated classes are almost certainly not due to genetic differences in the infants of these classes, to any inherent weaknesses in the infants born into poor families. The evidence, rather, indicates that the higher rates are due to the inadequacies, nutritional, sanitary, and the like, of the poorer infant's environment. The incidence and the mortality rates of such diseases as measles are not significantly different in the different socioeconomic classes. During epidemics the sickness and death rates of infants of higher socioeconomic classes are similar or tend to exceed those of the poorer classes. This suggests that there is no genuine difference in genetic susceptibility to disease in the infants of different socioeconomic classes.

The facts discovered about the growth of the school child, and its illnesses and mortality rates, in relation to socioeconomic environment are much the same as those for the infant and preschool

child. These facts were sharply brought out in one of the earliest studies made on the relation of socioeconomic factors to growth. This investigation, reported by Elderton, was conducted during 1905-1906 in Glasgow, Scotland, and involved the heights and weights of some seventy thousand children between five and eighteen years of age. The results obtained were then related to the types of school which the children attended. This yielded four district school groups of different socioeconomic ratings, from A to D, where A represented schools attended by children from the poorest districts and D those attended by children from the most prosperous. The results of this investigation are set out in Table IV.

From Table IV it will be observed that at practically every age, and for both sexes, an appreciable and regular difference was exhibited between the children of the four graded socioeconomic districts, in both height and weight, and this always in favor of the higher socioeconomic groups. For example, at age nine the average height of boys in district A was 48.8 inches, in B 49.5 inches, in C 49.9 inches, and in D 50.9 inches. At every age a steady increase is observed from group A to group D. Boys from district D at age five are 1.7 inches taller and at age thirteen are 2.5 inches taller than those of district A.

Later studies carried out in many different parts of the world have fully confirmed the capital importance of the environment and its effects upon the processes of growth. One of the most striking of these studies, made by Craven and Jokl, evaluates the growth records of 1,067 physically substandard adolescent boys at the Physical Training Battalion in Pretoria, South Africa. It was found that within the first nine months these boys spent in the training station they grew in bulk, on the average, at a rate five times as great as they would have grown in their unsatisfactory home environment. The factors in producing this remarkable acceleration in growth would appear to be mainly nutritional.

In Table V are set out the figures for five different English-speaking countries with somewhat different food and other habits, showing the percentages of children under average height, between the ages of five and fifteen years, divided by socioeconomic status. These figures tell a remarkable, and almost dramatic story. For one thing they show that the least number of children under

TABLE IV
HEIGHT (INCHES) AND WEIGHT (POUNDS) IN 70,000 GLASGOW (SCOTLAND)
SCHOOL CHILDREN, 1905-1906, BY ASCENDING ORDER (FROM A TO D) OF
SOCIOECONOMIC STATUS OF SCHOOL

Age:	5	6	7	8	9	10	11	12	13
<i>Boys</i>									
	<i>Height (inches)</i>								
Group A	41.3	43.0	45.1	47.0	48.8	50.6	52.3	53.8	55.2
Group B	42.1	44.0	45.9	47.7	49.5	51.1	52.8	54.3	55.5
Group C	42.1	44.0	46.2	48.1	49.9	51.5	53.5	55.0	57.2
Group D	43.0	44.8	46.9	49.0	50.9	52.6	54.2	55.9	57.7
<i>Girls</i>									
Group A	41.0	42.9	44.6	46.6	48.5	50.3	52.4	54.4	55.8
Group B	42.0	43.7	45.6	47.4	49.2	51.1	53.0	55.2	57.1
Group C	41.9	43.7	45.6	47.6	49.4	51.2	53.3	55.4	57.0
Group D	42.7	44.8	46.4	48.6	50.4	52.2	54.1	56.5	58.7
<i>Boys</i>									
	<i>Weight (pounds)</i>								
Group A	40.9	44.2	48.0	52.3	56.7	61.6	66.4	71.7	75.6
Group B	42.0	45.6	49.6	53.9	58.4	62.7	67.8	72.9	77.3
Group C	42.5	45.9	50.1	54.4	59.5	63.9	69.1	75.6	82.2
Group D	43.3	46.6	51.2	56.3	61.2	66.3	70.8	76.9	83.2
<i>Girls</i>									
Group A	39.9	43.0	46.4	50.5	54.7	59.5	65.3	72.4	76.8
Group B	40.6	43.9	47.7	51.8	55.8	60.8	66.8	74.3	81.3
Group C	41.3	44.7	48.1	52.7	56.9	61.9	68.4	76.1	83.0
Group D	41.8	45.6	49.3	54.3	58.8	64.4	70.5	78.8	89.0

SOURCE: E. M. Elderton, "Height and Weight of School Children in Glasgow."

average height in schools situated in prosperous districts were, in 1939, to be found in the United States, and this was true also of the average districts, but in the poorer districts the United States had a higher frequency of children under average height than the poor-district children of the other countries. The evidence indicates that in the year 1959 English children of all districts, because of the great care that has been spent on the nutrition of children since the beginning of World War II (1939), are seldom under average height.

TABLE V

PERCENTAGES OF CHILDREN, AGE 5 TO 15 YEARS, WHO ARE UNDER AVERAGE HEIGHT, CLASSIFIED BY COUNTRY AND SOCIOECONOMIC STATUS

Location of School	United States	Canada	Scotland	England	Ireland
Prosperous District	7.5	11.0	11.2	18.5	22.7
Average District	18.3	23.4	24.6	24.4	24.3
Poor District	36.4	31.6	27.3	35.1	30.7

Adapted from Cudmore and Neal, *A Height and Weight Survey of Toronto Elementary School Children 1939*.

Changes in Body Form with Changes in Geographic Environment

Beginning with Franz Boas's demonstration in 1912 that American-born descendants of immigrants undergo certain bodily changes, particularly in head form, a good number of similar studies on other groups have demonstrated even more fully how modifiable the form of the body is.

Table VI shows the kind and degree of differences between the measurements of immigrants' children born in the United States and those born in Europe. The results set out in this table prove that the form of the head may undergo certain changes with change in environment without change in descent. In other words, the pattern of the genotype may remain unaltered, but its physiological expression undergoes modification as a consequence of the effects exercised by a new environment. Furthermore, Boas showed that the influence of the environment makes itself felt with increasing intensity according to the time elapsed between the arrival of the mother and the birth of the child. This is well brought out in figure 12.

The American-born descendants differ in head form from their parents. The differences develop in early childhood and persist throughout life. The head index—that is, the head breadth taken as a percent of head length (cephalic index)—of the foreign-born remains practically the same no matter how old the individual at the time of immigration. This might be expected when immigrants are adult or nearly mature, but even children who

TABLE VI
INCREASE (+) OR DECREASE (—) IN MEASUREMENTS OF CHILDREN OF IMMIGRANTS BORN IN THE UNITED STATES COMPARED WITH THOSE OF IMMIGRANTS BORN IN EUROPE

Nationality and Sex	Length of Head (mm.)	Width of Head (mm.)	Cephalic Index	Width of Face (mm.)	Stature
Bohemians:					
Male	—0.7	—2.3	—1.0	—2.1	+2.9
Female	—0.6	—1.5	—0.6	—1.7	+2.2
Hebrews:					
Male	+2.2	—1.8	—2.0	—1.1	+1.7
Female	+1.9	—2.0	—2.0	—1.3	+1.5
Sicilians:					
Male	—2.4	+0.7	+1.3	—1.2	—0.1
Female	—3.0	+0.8	+1.8	—2.0	—0.5
Neapolitans:					
Male	—0.9	+0.9	+0.9	—1.2	+0.6
Female	—1.7	+1.0	+1.4	—0.6	—1.8

SOURCE: Boas, F., "Changes in the Bodily Form of Descendants of Immigrants."

come to the United States when one year or a few years old develop the head-index characteristic of the foreign-born. For Jews this index ranges around 83; that of the American-born Jews changes suddenly. The value drops to about 82 for those born immediately after the immigration of their parents, and reaches 79 in the second generation, that is, among the children of the American-born offspring of immigrants. The effect of American environment makes itself felt immediately, and increases slowly with the increase of time between the immigration of the parent and the birth of the child. Observations made by Boas in 1909 and 1937 yield the same results, save that there is an appreciable increase in all measurements in the 1937 series.

Similar confirmatory observations have been made on the de-

scendants of Puerto Ricans in the United States, on Russian Jews, Hawaiian-born children of Japanese, American-born Chinese, American-born children of Japanese, and American-born children of Mexicans.

What is the cause of such changes in the descendants of immigrants into the United States? We do not know. Is it nutrition? We cannot be sure. All that we can say is that the interaction between the developing genotype and the new environment has resulted in modifications of bodily form in the offspring of the immigrants. Once again, the importance of the environment is underscored.

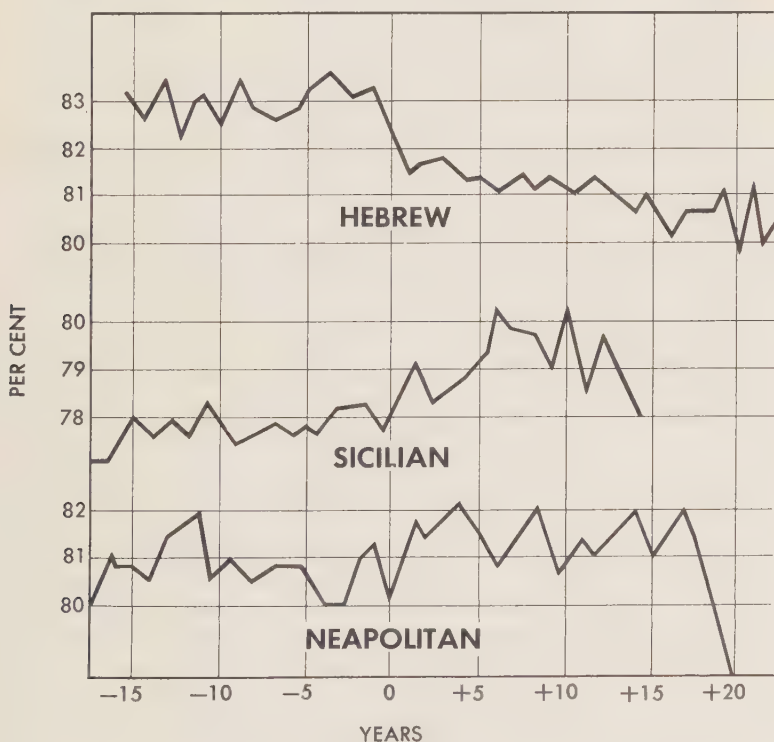


FIG. 12. Length-Breadth (Cephalic) Index of Immigrants and Their Descendants. (After Boas.)

Environment and Longevity

While there cannot be the least doubt that duration of life or longevity has a genetic basis, there can equally be no doubt that the environment plays a major role in calling the tune to which the genes will dance. The genes may have the capacity to dance all night, but it is the environment that decides, in interaction with the genes, how long the dance will be permitted to go on. An individual may start life with genes that would have been capable of maintaining him till he is eighty-five in a good environment, but a socioeconomically depressed environment, together with the psychosomatic (mind-body) changes which can afflict individuals in such environments, often makes it difficult for such an individual to attain half that age. On the other hand, an optimum environment enables many individuals to achieve a much longer life than would have been possible in a less favorable environment.

Perhaps the most spectacular effect of improved environmental conditions has been the increase in longevity during the last century. In 1858 the average duration of life in the United States was about 40 years. By 1900 this had jumped to 50 years; in 1920 this was 55 years, in 1930 a little over 60 years, and by 1958 the expectation of life for the average white American male was 67.4 years and for the average white female 73.6 years.* In Table VII the latest data are presented on longevity in forty-seven countries, comparing the latest available figures with those for the years around 1940. It will be seen from this table that the expectation of life at birth has increased appreciably throughout the world, the gains generally being largest in the countries which formerly had the least favorable record. In Puerto Rico, for example, the average length of life has increased more than 22 years in a fifteen-year period, from 46.0 years in 1939-1941 to 68.3 years in 1955. Even more striking is the record of Ceylon, where the expectation of life at birth rose from 42.8 years in 1946 to 59.9 years in 1954, an increase of 17 years in the short period of eight years.

*The expectation of life for nonwhites in United States by 1958 was 61.0 for males and 65.8 for females.

Mexico, Brazil, and Thailand have also made great gains, amounting to an increase in expectation of life at birth of about one year annually. These remarkable gains in longevity are undoubtedly due entirely to improvements in nutrition and sanitation, and in no little part to the magnificent work of the United Nations World Health Organization.

In identical twins, who are so called because they are derived from the same fertilized egg and are therefore genetically identical, environmental differences are undoubtedly responsible for the differences in longevity occurring among them. Thus, in Kallmann's study of twins who had lived beyond sixty years it was found that the difference in months between the age of death of 513 pairs of identical twins was 35.7, whereas in 1,226 pairs of nonidentical twins it was 73.7 months. These figures indicate at once the importance of the genes in the aging process and the duration of life, and the regulative effect of the environment upon those genes.

Environment and Mental Abilities

In an age of "I.Q.'s," "anxiety," "mental illnesses," "race differences," the feeble-minded and otherwise mentally retarded, "the Jukes" and "the Kallikaks," jeremiads on the declining national intelligence, and the fact that 1 out of 10 persons in the United States is mentally ill—in such an age, heredity and mental capacity have become closely linked terms in the minds of many people. The error most commonly committed is to identify the level of mental ability achieved by the individual with his genetic endowment. Everyone agrees that Mozart was a musical genius by virtue of his extraordinary genetic endowment and the training received in his home. No one doubts that had Mozart not been possessed of an extraordinary genetic endowment for music, no matter how thorough the training he received at home he would not and could not have become the prodigy that Mozart was. The genes had to be there first. Leonardo, Newton, Darwin, Einstein—all these men of outstanding genius were unquestionably genetically most extraordinarily well endowed. All they required was a minimal stimulus from the environment and their genius seemed to unfold itself almost in spite of themselves. Darwin regarded himself as a man of quite ordinary intelligence.

TABLE VII
EXPECTATION OF LIFE AT BIRTH, SELECTED COUNTRIES

Country and Period	Total Persons	Male	Female	Country and Period	Total Persons	Male	Female
<i>North America</i>				Ecuador (Quito)			
United States				1949-51	52.0*	50.4	53.7
1955	69.5	66.7	72.9	Venezuela			
1939-41	63.6	61.6	65.9	1946	47.0*	45.9	48.1
1955				1941-42	46.7	45.8	47.6
White	70.2	67.3	73.6	<i>Europe</i>			
Nonwhite	63.2	61.2	65.9	Austria			
Canada				1949-51	64.4*	61.9	67.0
1955	70.1	67.6	73.0	1930-33	56.5*	54.5	58.5
1940-42	64.6*	63.0	66.3	Belgium			
Costa Rica				1946-49	64.6	62.0	67.3
1949-51	55.7	54.6	57.1	1928-32	57.9*	56.0	59.8
El Salvador				Czechoslovakia			
1949-51	51.2*	49.9	52.4	1949-51	63.2*	60.9	65.5
Greenland				1929-32	53.6*	51.9	55.2
1946-51	34.9*	32.2	37.5	Denmark			
Guatemala				1946-50	68.9*	67.8	70.1
1949-51	43.7*	43.8	43.5	1936-40	64.7*	63.5	65.8
1939-41	36.5*	36.0	37.1	England and Wales			
Mexico				1956	70.5*	67.8	73.3
1949-51	48.7	46.7	49.9	1937	62.3*	60.2	64.4
1940	38.9*	37.9	39.8	Finland			
Puerto Rico				1951-55	66.6*	63.4	69.8
1955	68.3	66.7	70.0	1931-40	57.0*	54.5	59.6
1939-41	46.0	45.1	46.9	France			
<i>South America</i>				1954-55	68.2	65.1	71.4
Argentina				1933-38	58.7	55.9	61.6
1947	59.2*	56.9	61.4	Germany			
Brazil (Federal District)				1952-53 (East)	67.1*	65.1	69.1
1949-51	52.9	49.8	56.0	1949-51 (West)	66.5*	64.6	68.5
1939-41	42.5*	39.7	45.2	1932-34	61.3*	59.9	62.8
Chile				Hungary			
1952	51.4	49.8	53.9	1955	66.7*	64.7	68.7
1939-42	41.8	40.7	43.1	1941	56.6*	54.9	58.2

* Average of male and female.

SOURCE: Largely from United Nations Demographic Yearbooks and reports of various countries.

Einstein never ceased to wonder what all the fuss was about. Such responses on the part of men of genius to their own accomplishments confirm most people in the belief that ability is a matter of heredity, meaning genes.

Now, there is no doubt that genes and ability are fundamentally connected, but they are by no means the same things. Genes provide the potentialities, and it is the stimulus of the environment that brings those potentialities out. Even genius may fail to declare itself in the absence of the proper environmental stimulus. Paul Gauguin, the French painter, did not begin to paint until he was a mature man, and almost certainly would not have done so had he not lived in the very center of the painter's world. Grandma

TABLE VII
EXPECTATION OF LIFE AT BIRTH, SELECTED COUNTRIES

Country and Period	Total Persons	Male	Female	Country and Period	Total Persons	Male	Female
<i>Europe (cont.)</i>				<i>Africa and Asia</i>			
Ireland				Belgian Congo			
1950-52	65.8*	64.5	67.1	1950-52	38.8*	37.6	40.0
1940-42	60.0*	59.0	61.0	Ceylon			
Luxembourg				1954	59.9*	60.3	59.4
1946-48	63.7*	61.7	65.8	1946	42.8*	43.9	41.6
Netherlands				Cyprus			
1953-55	72.5*	71.0	73.9	1948-50	66.2*	63.6	68.8
1931-40	66.5*	65.7	67.2	India			
Northern Ireland				1941-50	32.1*	32.5	31.7
1950-52	67.1*	65.4	68.8	1921-31	26.7*	26.9	26.6
1936-38	58.5*	57.8	59.2	Israel (Jewish)			
Norway				1955	70.8*	69.4	72.1
1946-50	71.0*	69.3	72.7	1939-41	63.5*	62.3	64.6
1931-40	65.8*	64.1	67.6	Japan			
Poland				1955	66.1*	63.9	68.4
1952-53	61.4*	58.6	64.2	1935-36	48.3*	46.9	49.6
1931-32	49.8	48.2	51.4	Philippines			
Portugal				1948	51.2	48.8	53.4
1949-52	58.0*	55.5	60.5	1938	46.0*	44.8	47.2
1939-42	50.7*	48.6	52.8	Thailand			
Scotland				1947-48	50.3*	48.7	51.9
1956	68.6*	66.0	71.2	1937-38†	40.0*	36.7	43.3
1930-32	57.8*	56.0	59.5	Union of South Africa‡			
Sweden				1945-47	66.0*	63.8	68.3
1951-55	72.0*	70.5	73.4	1935-37	61.0*	59.0	63.1
1936-40	65.6*	64.3	66.9	<i>Oceania</i>			
Switzerland				Australia			
1948-53	68.6*	66.4	70.9	1946-48	68.4*	66.1	70.6
1933-37	62.7*	60.7	64.6	1932-34	65.3*	63.5	67.1
U.S.S.R.				Hawaii			
1954-55	64	61	67	1949-51	69.5*	67.8	71.3
1926-27‡	44.4	41.9	46.8	1939-41	61.0*	59.5	62.6
Yugoslavia				New Zealand‡			
1950	56.3*	54.5	58.2	1950-52	70.4*	68.3	72.4
				1934-38	67.0*	65.5	68.5

*Average of male and female.

†Bangkok municipal area.

‡European population.

Moses was an ordinary rural homemaker for the greater part of her life, until in her seventies she began to paint those delightful pictures she has given us. Had Gauguin and Grandma Moses lived in environments which failed to stimulate their desire to paint or failed to make it possible for them to do so, Gauguin might have remained a banker and Grandma Moses a pleasant old lady, and the world would have heard of neither of them.

Full many a gem of purest ray serene
The dark unfathom'd caves of ocean bear:
Full many a flower is born to blush unseen,
And waste its sweetness on the desert air.

Hackneyed as these lines may have become, we must still acknowledge their truth, and perhaps even agree that that truth has never been better stated. Very well, it may be said, but this truth does not alter the fact that genes and abilities are connected. This is true enough, but this is a very different thing from saying that genes *alone* determine abilities, for this they certainly do not. Genes did not determine Mozart's musical abilities. What determined Mozart's abilities was a combination of two things: (1) his extraordinary genetic endowment, and (2) the interaction of that genetic endowment with the appropriate environment. Had Mozart been brought up in a miner's hovel and from childhood been set to work in the mines without any musical exposure whatever, there would almost certainly have been no musical genius named Mozart.

But genius is an extreme manifestation of ability which is of rare occurrence in any generation of millions of human beings. Abilities at lower levels of achievement than genius appear to be even more dependent upon environmental stimulus than genius. Genius seems to require only the very slightest stimulus, and it is off at a bound. Handel as a child would find a spinet upon which to play even though forbidden to do so. Van Gogh, the Dutch painter, had only to discover his talent and in the few years that remained to him created his glorious canvases with a demonic fury in spite of an almost total lack of public appreciation. All that Edison needed was a workbench and the freedom to test the ideas that his world was waiting to see turned into practically useful things. But at the non-genius levels of ability human beings usually require a considerable assist from their environment and tend to realize their abilities in proportion to the amount of that assistance. Where human beings tend to receive little or no assistance from their environment in the development of their potentialities, there will be little or no development of them. Where individuals tend to receive a mediocre or moderate kind of assistance from their environment, their potentialities will tend to be correspondingly developed. Where human beings receive a high degree of assistance from their environment, their potentialities will tend to be most highly realized.

It is perhaps necessary once more to recall that environment means anything whatever that can act upon the individual. Bear-

ing this in mind we will remember that the environment of human beings tends to be quite complex in the number and quality of the different variables it involves. A child may receive the best schooling available in the land, but if its home conditions are emotionally disturbing, in spite of a perfectly adequate genetic endowment, that child may fail lamentably in schoolwork. On intelligence tests such a child may do very poorly. The damage he has suffered in his home environment may make it extremely difficult for him to recover his ability to learn for some years following an improvement in his domestic life. Such children are often puzzling. They do badly on intelligence tests; then on the tests of intelligence in everyday life they do well enough, but fail again very badly in their schoolwork. Often there is a reading disability, and should they tend to write backward and/or to read backward, this is often taken to constitute evidence that a genetic fault is somewhere involved. This may be so, and in some cases is in fact so, but it by no means necessarily follows that these disabilities are of a genetic nature. Nor does it necessarily follow that if the domestic environment of a child has been disturbing to it, it will fail at its schoolwork, or that the cause of failure if he does is definitely environmental.

Again, it must be emphasized that where human achievement is concerned causes are never exclusively genetic or exclusively environmental, but the result of the interaction of both. All that we are saying is that some children will respond very differently from others under the same kinds of environmental stimulation, and that unfavorable environments affect some children very much more unfavorably than they do others, and similarly that favorable environments affect some children very much more favorably than others. The differences in response are not necessarily principally due to differences in genotype—though in many cases this may be so—but may be due to a large extent to the environmental history of the individual since his birth—not to mention the substantial relevance of the prenatal history in many cases. Always, however, the resultant behavior is an expression of the interaction between the genotype and the environment. Hence, we must rid ourselves of the erroneous notion that ability is an exclusive expression of genetic endowment. It is *not*. Ability is the expression of the *interaction* between the genetic endowment of potentialities and the environments which the individual

experiences. Let us now proceed to consider the relevant materials.

Since this is an issue which has been much clouded by studies on "hereditary ability" or, at least, by the misinterpretation of the meaning of such studies, as well as befogged by certain famous studies on "hereditary disability," it is desirable to clear the atmosphere before proceeding further. In most such studies the common error is repeated of omitting to take into consideration the role played by the environment, and often what is mainly due to environment is credited to genes. The "goodness" of the genes that are transmitted in such families as the Russells, the Cecils, the Darwins, and the Wedgwoods cannot be doubted, but what can be questioned is whether any of the great men these families have given us would have achieved fame had they been born and raised in the slums. Favorable environments combined with their favorable genes enabled them to develop and express their abilities.

But what of those who occupy the other extreme of this spectrum of abilities, those who have practically no abilities or have mediocre abilities? Are they, in most cases, genetically deficient? That such persons *are* genetically deficient is a view which is widely held and supported by a number of famous works on the subject, the two most famous being those on "the Jukes" and "the Kallikaks." The subjects of these studies, the first published in 1875 and the second in 1912, have long been quoted as the horrid examples of the social and individual costs of "bad" heredity.

"The Jukes" and "the Kallikaks" are a euphonious combination of names that have become a synonym for depravity and degeneracy. Eugenists, criminologists, sociologists, and others have repeated the sorry story of these depraved families so often that they have come to enjoy not only a national but an international reputation. Yet the first are largely the creation of an unscientific, untrained, amateur criminologist, and the second have been saddled with a genetic history which, to say the least, is highly questionable.

"The Jukes" (the name is a pseudonym) were an extended family studied by Richard L. Dugdale in New York State in 1874. Dugdale seems to have had no preparation for such a study except that of interest, having recently become a New York Prison Association Inspector. Dugdale did not quite invent the

Jukes, but he certainly can be said to have compiled them, and compiled them after a method peculiarly selective quite evidently calculated to give support to his belief that there is a significant relation between crime and heredity. In fact, his naive assumption that crime is caused by heredity, "bad" heredity, is made quite explicit.

The difficulty with untrained investigators (and even with some trained ones) is that when they become enamored of a theory they tend to become insensible to the facts which are in opposition to it. To bolster his theory of the hereditary causation of crime, Dugdale fell back upon his imagination when the facts failed him. In his report, *The Jukes: A Study in Crime, Pauperism, Disease, and Insanity* (1875), Dugdale covers 7 generations, 540 "blood" relatives, and 169 related by marriage or cohabitation, harking back to the middle of the eighteenth century. Since many of the individuals involved had been dead for many years and information on them was hard to come by, Dugdale was forced to fall back upon such characterizations as "supposed to have attempted rape," "reputed sheep-stealer, but never caught," "cruelty to animals," "hardened character," and the like.

Altogether apart from Dugdale's naive assumption that crime, pauperism, disease, insanity, and other inadequacies are due to heredity, the inadequacies of his methods of inquiry and recording were so great that even in an age which was less deficient in reliable records than his own, his work would not be acceptable as throwing any light upon the causation of those conditions. Socially inadequate lineages undoubtedly exist, but even as a representative of such a lineage Dugdale's pedigree of the Jukes is so full of assumptions and prejudices that it cannot be used for any purpose other than to provide an example of the manner in which such studies should *not* be made.

Were reliable pedigrees of socially inadequate lineages available the problem presented by them would be not to prove them as being due to either heredity or environment but to discover, if possible, what roles each of these factors actually played in the production of the social inadequacy in each individual.

Forty years after the original study of the Jukes, they were the subject of a follow-up study by another investigator wedded to the "heredity" theory of the causation of crime, A. H. Estabrook, whose study was published in 1916. He found that there

were still a good many "inadequates" among the Jukes, but surprisingly enough he also found that there were a fair number of decent respectable citizens, some even "superior," and others who had done quite well in the world. This was attributed to the fact that they had married outside the Jukes clan and received new infusions of "better" genes.

"The Kallikak Family" (a fictitious name formed from the combination of the Greek words meaning "good" and "bad") is the other famous family. The Kallikaks were studied by Dr. Henry H. Goddard, the director of the Vineland Training School for the mentally retarded at Vineland, New Jersey. There were two clans of Kallikaks, the "good" and the "bad." The "good" and the "bad" ones were both descended from the same Revolutionary War soldier, Martin Kallikak, but from different women with whom he had formed unions. His first union, it is alleged, was with a feeble-minded girl, who gave birth to Martin Kallikak, Jr., who was so bad that he became generally known as "Old Horror" and in turn became the father of no less than ten other "horrors," from whom all the other hundreds of horrible Kallikaks traced by Dr. Goddard were descended. The hundreds of "good" Kallikaks were, of course, descended from Martin's marriage with an estimable Quakeress.

As Scheinfeld and others have pointed out, if "Old Horror" was a degenerate largely or entirely because of his genes, then it is clear that no single dominant gene could have been involved. So complex a condition as "social inadequacy" or "degeneracy" could not be due to a single gene, and must therefore in genetic terms involve at the very least a pair of recessive genes. This being the case, then the estimable Revolutionary soldier Martin Kallikak, Sr., must have carried duplicates of the defective genes situated in the chromosomes of the feeble-minded girl who gave birth to their alleged son who subsequently earned notoriety if not fame as "Old Horror." In which event it follows that something passing strange happened in the lineage of the "good" Kallikaks, for by some extraordinary piece of good fortune not a one of them seems to have inherited a pair of "bad" recessive genes for "degeneracy." If genes are involved this is what should have occurred in some of them, for if Kallikak, Sr., was the father of the feeble-minded girl's child then he proved himself a carrier of defective recessive genes. In which case these should have been

transmitted to some of his descendants, and some of these descendants would almost certainly have married individuals with similar genes, and a certain number of their offspring should, then, have shown "degenerate" traits. But they didn't. What, then, are we to conclude? That the "good" environment of the "good" Kallikaks was sufficiently strong to overcome the expression of their defective genes? Or that Kallikak, Sr., has all these years innocently borne this blot upon his escutcheon of the paternity of a child he never sired? Or did the "good" Kallikaks just by chance manage to avoid picking up any defective recessive genes? Or are they possibly a case of paramutation? The reader may be left to decide for himself.

The Jukes and the Kallikaks are often quoted in books and articles as examples of what a "good" and a "bad" heredity can do to human beings and society. But no reputable scientist who has any acquaintance with the facts of genetics and its methods considers that such studies belong to anything but the category of the recklessly quaint anecdotal method of investigation. The complete disregard by these early investigators of the possibility that the effects they described might have been due to environment rather than to genes, renders their work valueless except for the fact that it demonstrates that extreme poverty and undesirable social traits can be *inherited through the environment* by successive generations. The Nobel prizewinner and great pioneer in the study of heredity, Thomas Hunt Morgan, categorized such studies very properly when he said, "The pedigrees that have been published showing a long history of social misconduct, crime, alcoholism, debauchery, and venereal diseases are open to the same criticism from a genetic point of view; for it is obvious that these groups of individuals have lived under demoralizing social conditions that might swamp a family of average persons. It is not surprising that, once begun from whatever cause, the effects may be to a large extent communicated rather than inherited."

And that is the point. It is quite possible that a genetic defect may have been involved in some of these pedigrees, but that was not demonstrated. The fact that a family line may for generations live in poverty and exhibit in many of its members undesirable traits of various kinds tells us nothing about the genetic endowment of those individuals. All that we know is that they have lived for generations in a "bad" environment, and we know that "bad"

environments have a way of making it difficult for people to extricate themselves from them.

The particular social environment of the individual, his *cultural environment*, adds a fourth dimension to what the individual inherits, a new zone of adaptation. Since the individual acquires most of his mental traits from his cultural conditioning, and since this is solidly accomplished by the time he reaches his seventh or eighth year, he thereafter carries his cultural, his behavioral, environment around with him wherever he goes. His cultural conditioning virtually obscures whatever role the genes may have played in the development of his mental traits. It is this that makes it so difficult to discover what may have been due to genes and what to cultural conditioning. Hence, studies such as those of the Jukes and the Kallikaks are much worse than worthless, for they confuse the issues, make appear easy what is difficult to study, and with an easy facility assert conclusions which cannot be substantiated by the evidence upon which they are supposed to be based. Finally, by attributing to "heredity," to genes, what may have been either wholly, or certainly in part, due to environment, such studies have served to misplace the emphasis and to direct our attention in the wrong direction.

In the passages that follow the intention is to show that no matter how well- or ill-endowed the individual is, he is likely to realize his potentialities, his abilities, only to the extent permitted by the environment with which he interacts. And this is, for the most part the *cultural environment*. We had better describe more fully what the cultural environment is.

By *culture* the anthropologist means the man-made part of the environment, everything that the individual learns—the way of life of a people, its pots, pans, institutions, mores, beliefs, religion, social organization, educational practices, ways of bringing up children, occupations, and the things that are expected of human beings at their different ages, grades, and in their different statuses and roles.

Numerous studies have shown that culture and ability are closely connected with one another. For example, persons living in different cultures often differ as a group in the development of certain abilities from the members of other cultural groups. Similarly, the different social classes within the same culture will often exhibit differences in ability. In the first instance such dif-

ferences are often attributed to "racial" factors. In the second instance the differences are quite as frequently attributed to "hereditary" factors. This is, of course, an easy way to deal with such differences. All one need do is to say that they are either due to "race" or to "heredity," and there we are, absolved from all responsibility of really doing the work of discovering what the true answer may be. Let us see what happens when we try to discover the truth.

It has long been well known that American Indians do not do well on intelligence tests. It has sometimes been said that this might be due to the fact that the intelligence tests we give them are foreign to their ways of thinking, to their cultural conditioning, that therefore we should not expect them to do as well on the tests as persons who belong to the culture for which those tests were devised. This is what the experts think, but others have preferred to think that American Indians simply are not as intelligent as whites. Let us take a case in point.

The average American Indian obtains an I.Q. score of about 80. This is when he is living on his own reservation among his own people and has adopted as little as possible of the ways of the whites. Some years ago oil was found on the reservation owned by the Osage Indians of Oklahoma. This enabled the Osages to improve their economic and social conditions very considerably and to bring them more in line with those existing among the whites. In a study by Rohrer, it was found that Osage children when given the Goodenough "Draw-a-Man" test, scored an average I.Q. of 104 while the white children scored 103. On a verbal I.Q. test the Osage children obtained an average score of 100 while the white children obtained a score of 98—the difference between the Indian children's score and that of the white children was insignificant. It showed that when the environmental opportunities are more or less equalized for Indian and white children, the Indian children can do at least as well as the whites.

American Indian children placed in white foster homes were found to have an average I.Q. of 102. Brothers and sisters of these children still living on the reservation were found to have an I.Q. of only 87.5. The difference in favor of the foster-home children was undoubtedly due to the improvement in their environmental conditions.

It is widely believed that the I.Q. test measures "inborn intelli-

gence." This is an error. What the I.Q. test measures for the most part is the response that the individual is able to make to the tests as a consequence of his experience of life. Though his genetic potentialities may be involved to some extent, often the experience of the tested individual has been of such a nature as to make it difficult for him to get anywhere near his potentialities. Children in our own culture may be so confused and upset by their life experiences as to make it extremely difficult for them to do well by their potentialities. Cross-culturally, that is to say, as we cross from one culture to another, ways of thinking about and perceiving the world become so different that tests designed in one culture are hardly applicable to the members of another. If the natives of New Guinea or of any other nonliterate people were to apply *their* I.Q. tests (supposing they had any), we should not, I am afraid, do very well by them.

When Negro children migrate to New York City or to Philadelphia after some years of residence in the South, their I.Q. scores undergo a gradual elevation until they are only slightly below those of the white children in those cities. But they are way above those of the Negro children of their own age who have remained in the South. The improvement in the conditions of the environment in the North, as compared with those under which the Southern Negro children live, is almost certainly responsible for the improvement in the I.Q. scores of the Negro children who have migrated from the South.

Negro and white babies tested during World War II at New Haven were found not to exhibit any significant differences in physical or psychological development. With good wages, and improved working conditions, the Negro parents were able to supply their infants with all that was required to equalize their conditions with the white babies.

Similarly, children who move from rural to urban environments on the average achieve a higher I.Q. than those who remain in the rural environment. On the whole urban-dwellers do better on intelligence tests than rural-dwellers, though the difference in their scores has become increasingly reduced in recent times. In earlier days rural areas were not as well equipped with schools as they are today; television and other modern communications were not as available as they are today. Hence, with the improvement in the environment rural-dwellers have undergone a cor-

responding improvement in I.Q. Such tests, however, are still devised by urban-dwellers and contain more items with which the urban-dweller is more familiar than the rural-dweller. When tests were devised that were based on rural experience and administered to city and rural children, the children from the city did not do as well on the tests as the children from the rural areas. The opinion of Dr. Myra Shimberg, who applied this test, is that it was no less fair to the city children than the standard test is to the rural children, and that neither test sanctions any conclusion as to innate intelligence.

As an illustration of the manner in which response to intelligence tests is conditioned by environmental experience, we may cite the experience, reported by Pressey, of an intelligence tester among "poor white" children of the Kentucky mountains. The question was asked: "If you went to the store and bought six cents' worth of candy and gave the clerk ten cents, what change would you receive?" One child immediately responded, "I never had ten cents and if I had I wouldn't spend it for candy, and anyway candy is what your mother makes." The examiner tried again: "If you had taken ten cows to pasture for your father and six of them strayed away, how many would you have left to drive home?" Whereupon the reply came forth, "We don't have ten cows, but if we did and I lost six, I wouldn't dare go home." The examiner made one final desperate effort: "If there were ten children in a school and six of them were out with measles, how many would there be in school?" Without hesitation the answer came back, "None, because the rest of them would be afraid of catching it too."

These were highly intelligent answers, but they were conditioned by the child's experience and not by that of the examiner. The same point is amusingly illustrated by the answers given an intelligence tester who asked a little boy who was accustomed to wearing a sombrero, "What would happen if I pulled your hat over your eyes?" "I couldn't see," replied the boy. "Now," said the examiner, "what would happen if I cut off your ears?" "I couldn't see," was the prompt reply. "You couldn't see?" exclaimed the examiner quite incredulously. "Yes," said the boy. "My hat would fall over my eyes." This reply was, of course, entirely accurate, and a great deal more so than the reply the examiner was expecting, namely, that the boy wouldn't be able to

TABLE VIII
MEAN TEST SCORES OBTAINED BY 16-YEAR-OLDS BY ASCENDING ORDER
(FROM A TO D & E) OF DIFFERENT SOCIAL-STATUS GROUPS

Social Status	No.	Stanford-Binet I.Q.	Wechsler-Bellevue Perf. I.Q.	Iowa Silent Reading Score	Paper Form Board	Minn. Mech. Assem. T-Score (Boys)	Chicago Mech. Assem. T-Score (Girls)
A	13	98	103	45.6	31	53.0	45.3
B	49	104	102	48.9	31	48.8	48.5
C	44	112	109	51.0	40	51.6	52.0
D & E	9	128	118	58.0	44	46.8	62.1

Adapted from Janke, L. L., and Havighurst, R. J., "Relations Between Ability and Social Status in a Midwestern Community, II. Sixteen-Year-Old Boys and Girls."

hear—which would have been wrong, though the examiner would have thought it the right answer to the question.

The I.Q. test is a test of an actuality, not of a potentiality. One can only get significant meaning out of an I.Q. test when the environments of the subjects have been reasonably equal. Between socioeconomic classes a correction should always be made for differences in environmental experience.

Class differences in intelligence are as striking for children as they are for adolescents and adults. Class differences in intelligence express themselves as significantly in three-year-olds as they do in eighteen-year-olds. This has sometimes been taken to indicate that hereditary differences must exist between the classes, that those who have what it takes tend to rise in the scale, and that naturally such genetically superior individuals tend to have genetically superior children. This does not necessarily follow, however. Parents who have achieved a middle- or upper-class status need not have done so because of superior genes, but because of superior opportunities. The fact that gifted children come proportionately more often from families in which the father is a professional man than from families in which the father is a member of the skilled-labor class does not necessarily mean that significant genetic differences exist between the profes-

sional classes and the skilled laboring classes. Such differences may exist, but we have no definite evidence of this. On the other hand, it could just as well be true that there are no significant differences in the distribution of genes for ability between these classes, but that there have been very significant differences in the kinds of opportunities which the members of these classes have had to develop their abilities.

That environmental experience plays a major role in test results is demonstrated by the fact that achievement on these tests varies with the nature of the test, although on nearly all tests the tendency has been for the score to rise with the rise in social class. For example, in tests of sixteen-year-old boys and girls it was found that the boys of the lowest classes on the whole did better on a mechanical assembly test than the boys of the upper classes. The explanation offered for this is that the boys of the lowest classes had probably had more experience with mechanical things than the upper-class boys. Interestingly enough, the girls of the upper classes achieved a significantly higher mean score on this same test than the boys of any of the social classes!

In tests of primary mental abilities administered to thirteen-year-olds, the tests vary even more markedly for the classes with the function tested. Number, verbal comprehension, and word fluency show bigger differences between the classes than do the tests for space, reasoning, and memory. The strong suggestion is that there is a significant relationship between the test results on each of these items and social or cultural environment. As might have been expected it has been found that children of the higher classes are much better at tests involving a knowledge of academic or bookish words than are children of the lower classes. Thus, tests may themselves be, and indeed often are, culturally biased. When the tests are put in simpler language the responses markedly improve among those for whom the more bookish words have no meaning.

Twins reared apart are extremely interesting with respect to environment in relation to genes, for since identical twins are of the same genetic structure we have in those who have been reared apart an excellent means of discovering to what extent different environments are capable of influencing the expression of the genes. But this is a subject which deserves a chapter to itself.

Twins, Genes, and Environment

MULTIPLE BIRTHS, throughout the world, adhere closely to the theoretical expectations. They are as follows:

- Twins occur in 1 out of approximately 87 births
- Triplets occur in 1 out of approximately 7,569 births
- Quadruplets occur in 1 out of approximately 658,507 births
- Quintuplets occur in 1 out of approximately 57,289,761 births
- Sextuplets occur in 1 out of approximately 4,984,209,207 births

There is an apparent mathematical relationship between the different kinds of multiple births, for triplets occur as the square (or second power) of the number of twin births; quadruplets occur as the cube (or third power) of the number of twin births; quintuplets occur as the fourth power of the number of twin births (or the square of the number of triplets); and sextuplets occur as the fifth power of the number of twin births.

There are only about fifty authentic records of quintuplet births, and of sextuplet births there are less than five authentic records.

For about every 95 deliveries in the United States 1 set of twins is born. This means that about forty thousand sets of twins are born each year in this country. About thirteen thousand of these sets are identical twins. There are two kinds of twins: *identical* (monozygotic = one-egged) and *fraternal* (or dizygotic = two-egged) twins. Identical twins develop from the same fertilized egg and are always of the same sex and contain the same set of genes, and so closely resemble each other that they are hardly distinguishable. Fraternal twins develop from two separate fertilized

eggs and may be of the same or opposite sex, and resemble each other no more closely than do brothers and sisters born at different times. A little over one fourth of all sets of twins born are identical; the other three fourths are fraternal.

Because identical twins are genetically identical they afford us an opportunity to study the relative effects of genes and environment in interaction with one another. Everyone knows of at least one set of twins who have been raised in the same family, dressed alike, treated alike, and who so closely resemble one another that even those closest to them sometimes experience difficulty in distinguishing which is which. Such cases tell us that individuals with the same genotype when raised in the same environment will make much the same responses to it. But they do not tell us what would happen if these identical but separate individuals were raised in different environments. This is something about which we can learn by studying those identical twins who have been separated from one another since early infancy and have been raised in different environments. The classic study on *Twins* by H. H. Newman, F. N. Freeman, and K. J. Holzinger, published in 1937, gives a detailed account of nineteen pairs of twins who were



FIG. 13. Sextuplets. Five boys and a girl born to a West African woman April 19, 1903, at Accra, Gold Coast. The girl and one boy had a placenta each. The remaining four boys were attached by pairs to two placentae. One boy died two days after birth, four boys died three days after birth, and the girl four days after birth. On her first confinement the mother gave birth to quadruplets, on her second and third to triplets. Thus, in four confinements this woman gave birth to sixteen children. (From H. Cookman.)



FIG. 14. Four Sets of English Quadruplets. (Courtesy of *Family Doctor*, London.)

so separated. Since 1937 a fair number of other studies have been published on smaller series of identical twins, and all agree in finding that there are both remarkable likenesses and remarkable differences between identical twins reared apart.

In spite of differences in their separate places of residence the twins of these studies, who lived in America, were on the whole in much the same physical environment and experienced much the same nutritional history; hence, as might have been expected, in physical appearance, in height, and in weight, they maintained the closest resemblance to each other. The exceptions to this were in the cases where one twin had developed a more or less severe illness and the other had not. There have been several cases recorded where one identical twin was markedly taller than the other and heavier in spite of the similarity of the environment in which they were raised (see fig. 16). Differences in identical twins have also been recorded for their ability to taste certain substances, susceptibility to disease, and fecundity or the ability to bear children. But on the whole everyone is impressed by the remarkable

physical and psychological likenesses that exist between identical twins, even between those who have been separated from infancy. However, the differences that *are* found to exist between identical twins must be due to environment, since the twins are genetically identical. Hence, we arrive once more at the general rule that identical genotypes in different environments will respond in accordance with the differences set by those environments. All investigators agree that some traits are more susceptible to environmental influences than others; such traits, for example, are height, size, shape, dimensions of the head, and scores on the test for neurosis (Woodworth-Mathews test).

In the series of nineteen sets of twins studied by Newman and his colleagues, except for six of the sets, there were no more significant differences between the separated pairs of twins than they



FIG. 15. The Diligenti Quintuplets. (Courtesy of *Family Doctor*, London.)

found to exist among unseparated pairs of twins. This strongly suggests the power of the genes and the limitation of the effect of environment. But in this connection it must be remembered that although the identical twins of the separated groups lived in different families far removed from each other, nevertheless the environments in those families were not, on the whole, substantially dissimilar. Usually the twins were separated as a result of the mother dying at or shortly after birth, whereupon one twin would be adopted by a member of the family and the other twin would be adopted by another member of the family, or one or both twins might go to an institution for a time and then be adopted by different families in different parts of the country. In every case the effort would be made to put each child in a similar home of a class background similar to that of its own family. Therefore, it should not be surprising to find that nonseparated and separated sets of identical twins do not differ markedly from one another in the development of any of their traits. The genes in each set being the same, and the environments for most sets being similar, one would not expect to encounter any markedly dissimilar developments. But in those cases in which there has been a more substantive difference in the environments of the separated twins, the differences between them, especially involving those traits which are more susceptible to environmental influence, are more substantial.

To commence with, let us describe the case history of a set of separated identical twins which throws a rather astonishing light upon the "strength" of the genes.

Ed and Fred were seen by Newman when they were twenty-six years of age. They were separated in very early infancy and were adopted by two different families, of essentially the same socioeconomic status, living in the same New England town. They were each brought up as only children. They went to the same school for a time but never knew they were twin brothers, even though they had noticed their remarkable resemblance. When they were about eight years old, the two families both moved away and became permanently separated. The boys did not meet again until they were twenty-five. Edwin lived most of his life in a large city in eastern Michigan and Fred in a medium-sized city in western Iowa. On the whole there seems to have been no marked differences in their social environments.

Edwin learned of his twin's existence when he was repeatedly mistaken for his brother. Having informed his foster parents of

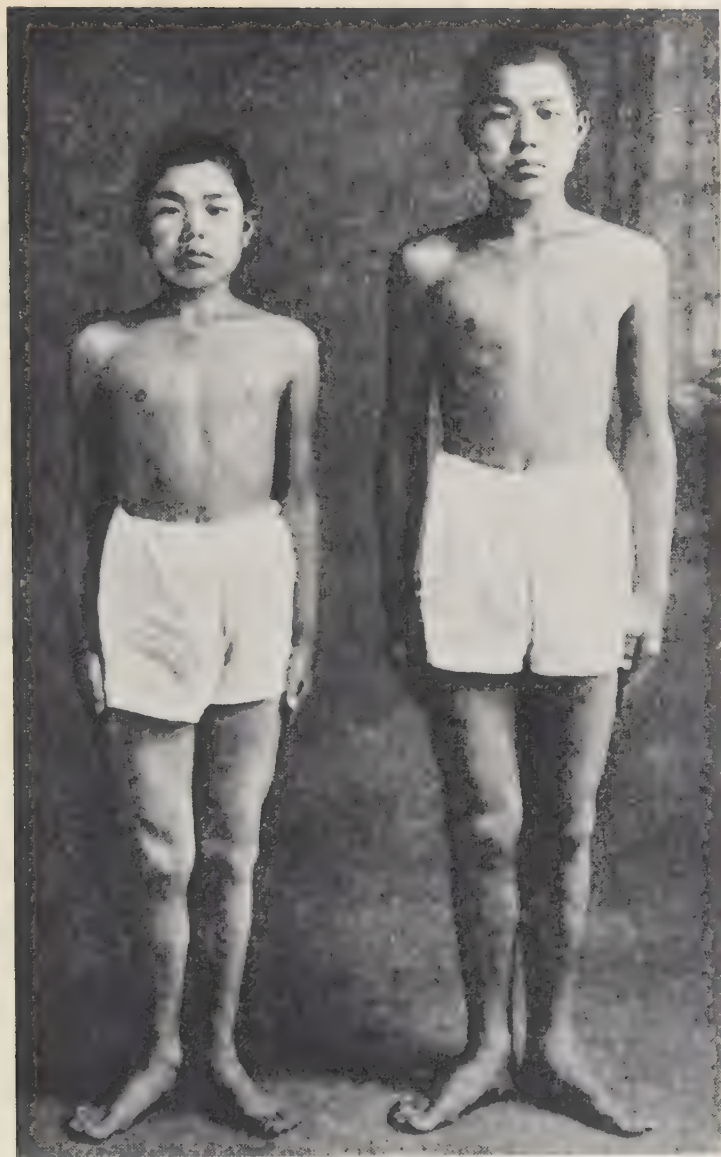


FIG. 16. Difference in Stature of Identical Twins. (From Komai and Fukuoka.)

these incidents they then told him that he did have a twin brother. Arrangements were made for the pair to meet, and when they did so the following facts emerged about them.

Edwin and Fred looked as like each other as identical twins reared together. Edwin was a half-inch taller than Fred and weighed one and a half pounds more. Eye color, hair color and form, hairline, beard, complexion, ears, features were virtually identical. The teeth were irregular in both in the same way. In both the upper middle incisors were turned inward in the midline. Each had a supernumerary upper eyetooth placed too high in the gum, and Edwin had had this extracted. The extra tooth was on the right side in Edwin and on the left in Fred—a case of mirror-imaging. The hair whorls, however, ran clockwise. Both were right-handed. General body build and carriage were the same in both.

Though they had been so long and thoroughly separated from one another, not being aware, indeed, of each other's existence, Edwin and Fred had led remarkably parallel lives. Both had been reared as only children, both had had about the same amount of education. They had each developed an interest in electricity, and both had become expert repairmen in branches of the same telephone company in their respective cities. They married young women of about the same age and type, each in the same year. Each had a baby son, and each—believe it or not—owned a fox terrier whom they called by the same name, Trixie!

Make what you will of this astonishing story, the facts are as stated. Given environments as close as those Edwin and Fred lived under and given their identical genetic background, astonishingly similar as their independently lived lives were, the likenesses are perhaps understandable. Given the same genotype and a similar environmental experience in two separated individuals, one may reasonably expect similar responses from them.

In behavior and personality there was a striking basic similarity, though Edwin was the more flexible, emotional, and easily aroused. He was more vivacious and responsive than Fred, possibly due to some difference in their environment. Edwin appears to have had better and more continuous schooling than Fred, though both were poor at their studies. However, Fred was the better speller, his handwriting firmer; but even so there was a good deal of similarity in their handwriting.

Edwin and Fred illustrate what happens when identical twins though separated grow up in similar environments. Now let us cite a case illustrating what happens to identical twins when they are brought up in contrasting environments. Such a case is that of Gladys and Helen.

Gladys and Helen were first seen when they were thirty-five years old. They were born in a small Ohio town and were separated when about eighteen months of age. They did not meet again till they were twenty-eight years old. Helen had been twice adopted. In the first instance the foster father turned out to be an unstable person and the foster mother became mentally ill two years after Helen had been adopted. Helen was therefore taken back to the orphanage, and after several months, was again adopted by a farmer and his wife who lived in southeastern Michigan. This was her home for the next twenty-five years. Her second foster mother, though she had had few educational advantages herself, was determined that Helen should receive a good education. So Helen eventually graduated from college. She taught school for twelve years, married at twenty-six a cabinetmaker with a high-school education, and had a daughter. In the course of her education she had acquired a great deal of polish, showed much ease in social relationships, and was possessed of considerable feminine charm.

Gladys had been less fortunate. She was adopted by a Canadian railroad conductor and his wife. When she was in third grade her foster father was stricken with an illness which necessitated his removal to a rather isolated part of the Canadian Rockies. Since there were no schools available Gladys's formal education came to an end and could hardly be said to be resumed when the family returned to Ontario. So she stayed at home and did housework till she was seventeen and then went out to work in a knitting mill. At nineteen she went to Detroit and worked as a saleswoman in stores and did some clerical work, finally getting a job in a small publishing establishment where she became assistant to the president. Gladys married when she was twenty-one, her husband being a mechanic with a high-school education.

Helen was healthy as a child and had no serious illness as an adult. Gladys was very delicate as a child, had several serious illnesses, including scarlet fever, very nearly dying of measles. Both had quarrelsome foster parents. For the rest, their environments

had been very similar, except in relation to the educational experience.

Physically Helen looked her age at thirty-five, but Gladys looked about forty. As for the remainder of their traits they were physically highly similar. Helen weighed 140 $\frac{3}{4}$ pounds, and Gladys 139 $\frac{1}{4}$ pounds. Helen was 62.1 inches in height and Gladys 61 inches. Gladys had broader shoulders and was more mannish in figure and carriage than the more feminine Helen. Hair color and form were the same, except that Helen's hair was a little grayer. Eye color was the same. Teeth were white and regular—each had six fillings of cavities affecting the molars and premolars. Thus, physically they were very much alike. The differences that distinguished them, differences in carriage of body and facial expression were obviously associated with the different social lives they had led.

Helen was confident, suave, graceful, made the most of her personal appearance, and was the more overtly aggressive. Gladys was diffident, ill at ease, staid, and stolid. She was without charm or grace of manner. She made no effort to make the best of her physique or to create a favorable impression. As Newman and his colleagues remark, "As an advertisement for a college education the contrast between these two twins should be quite effective."

On all mental ability and scholastic tests Helen showed marked superiority to Gladys. Helen scored an I.Q. of 116 and Gladys an I.Q. of 92, a difference of 24 points. We have already seen that there were wide differences in personality traits. The styles of handwriting were very different, Helen's being mature, Gladys's, that of a fourteen- or fifteen-year-old child, although there was a marked similarity in which both wrote a rather peculiar letter *f*.

Considering the nature of the environmental experiences of these identical twins reared apart, one whose formal education virtually stopped at the third grade, the other who went through high school on to college from which she graduated with a B.A. degree, the differences observed in Helen and Gladys as phenotypes are not surprising. Their case again underscores the fact that what we can do is set by the genes, but that what we actually do is largely determined by the environment.

Attempts have been made by various writers to show that in spite of considerable differences in the environment of twins who have been separated for many years, they have nevertheless in vir-

tually all respects remained closely similar. When, however, one comes to examine the cases these writers cite, it is found that the separation occurred as late as adolescence, or even later, and furthermore that the differences in the twins' environments were not really as great as they superficially appeared to be. For example, Dr. Franz J. Kallmann cites the case of identical twin sisters whose life history "approximated the widest possible discrepancy imaginable in our culture. It would be difficult to find twin partners separated by a greater distance and under more varied social, cultural, and climatic circumstances, or exposed to greater differences in dietetic and personal living conditions than were these twin sisters, now 85 years old." Both were raised in a small rural community. One sister married a local farmer at age eighteen, the other entered a Bible school in her middle twenties, after which she went as a missionary to the Orient, where she remained until she rejoined her widowed sister at the age of sixty-five. After forty-seven years of separation it was difficult to tell the twins apart, they were so much alike. The married twin who had had six children was twenty-eight pounds heavier, arrived at menopause seven years after her sister at the age of fifty. Her vision was slightly more impaired, and she did not score quite as highly on the intelligence test, though she was of superior intelligence.

These twins were separated when they were between eighteen and twenty-one years of age. Up to that time they had lived in the same home, their basic personalities had already been formed and their basic education completed. It is not surprising that they should have remained as much alike in personality traits and abilities as they did. As for the "widest possible discrepancy" in their life histories, this obviously refers to the period after their separation, a period which insofar as the development of psychological or behavioral traits are concerned is in no way to be compared with the importance of the first twenty years of life; anything that happens thereafter, alas, is, as most of us know, not nearly as important to the formation of our basic character. Furthermore, in spite of the differences in cultural milieu and diet, the missionary sister probably spent the greater part of her time living in a mission with her own kind, so that the environmental differences between the twins were probably by no means as great as might be supposed. In any event, it is well known that regardless of differences in environment following adolescence, that is, when physi-

cal and psychological growth has been virtually completed, twins tend to age in a remarkably similar manner.

Since psychological traits depend so much upon experience, it is to be expected that significant differences in experience, in life history, will be reflected in those traits. On the other hand, traits that are not as susceptible as psychological ones to variations in the environment, such as hair color and form, eye color, form of features, teeth, and the like, are more likely to show a high degree of similarity in identical twins. To show the extent to which such identity may be realized in twins I shall refer to a study I carried out on the dentition of a set of identical twins. This study is somewhat unusual since it involved a mathematically and geometrically exact analysis of the position of every cusp on each tooth in both the upper and lower jaws and the exact location of these cusps compared in both twins.

The Dentition of a Set of Identical Twins

These identical twin boys were selected at random in order to discover how closely their teeth resembled each other. The twins, whom we shall call Daniel and George, were studied over the course of several years. They were white Americans. George was born first, Daniel a few minutes later. George has always been bigger than Daniel. George was born with an enlarged heart and has always had a left auricular systolic murmur. Daniel is left- and George right-handed. Physically and temperamentally they were as alike as could be, except that, according to the father, George was more reasonable and tactful than Daniel, while Daniel was more truthful than George. First examined when they were 6 years 1½ months of age, the detailed measurements of the bodies of these boys proved that they were astonishingly alike. Examination of the teeth revealed striking likenesses in every respect, but what was most interesting was that a complex identical pathological condition had developed in both boys, involving the same teeth.

This remarkable pathology consisted of a marked resorption of the furthest roots and a portion of the crown of the second molar of the milk teeth on the right side of the upper jaw. This resorption had, at least in part, been produced by the pressure of the descending first molar of the second or permanent set of teeth. This had grown downward and forward into the milk tooth, its nearest

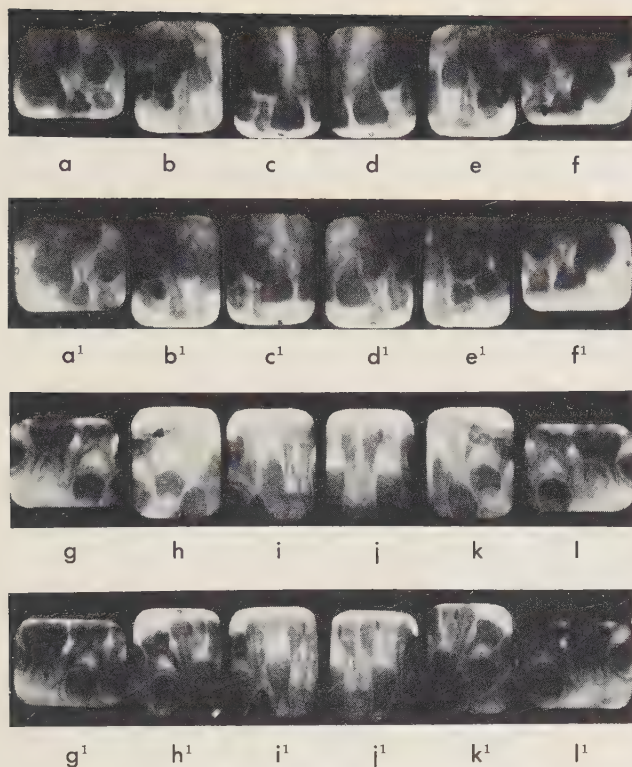


FIG. 17. (1) X rays of the teeth of the upper jaws of identical male twins aged 6 years and 1½ months showing identical pathology of first permanent molar eroding into the second milk molar, f, f¹. (2) X rays of teeth of lower jaws in same identical male twins aged 6 years and 1½ months. (3) A mathematically exact projection of the teeth of same identical male twins aged 7 years and 11 months. Left, upper jaws; right, lower jaws. (From Ashley Montagu.)

portion lodging in the excavated basin of the milk tooth. This may be clearly seen in the X rays of the teeth shown in figure 17. Such a pathological condition would be remarkable as an occurrence in any individual; its exact duplication in two separate individuals is little short of amazing. As we can see from the X rays the state of development of the teeth is virtually identical in both twins. Their genes being identical and their environment as nearly similar as possible, it is not surprising that even a complex exaggeration of the normal eruption of a permanent tooth in relation to a milk tooth should follow a similar course in each twin.

In this case we have an illustration of the fact that where the genotypes are the same and the environment is as nearly as possible the same, the responses made by the organism, as a result of interaction between genotype and environment, will be the same.

Quite as remarkable in these twins is the virtually identical manner in which the teeth have erupted and are placed within the jaws (see fig. 17, d-g). The cusps, grooves, ridges, edges, and the centroids of each tooth are almost indistinguishable, except for the right lateral incisor of the upper jaw, which has undergone a slight rotation in George. At the time the map shown in d was made, Daniel had lost his two upper central incisor teeth. George lost his five weeks later.

When it is considered that there are twenty teeth in the milk dentition and thirty-two in the permanent dentition, and that each of these teeth can erupt in any number of different ways, not to mention the number of possible variations in form, the chances of any two series of dentitions bearing an identical resemblance to each other in any two separate individuals are astronomically remote. In fact, the chance of getting any two like combinations in man is determined, on a purely numerical basis, by the number of his chromosomes, so that in the mating of two individuals each with a haploid set of twenty-three chromosomes, the chance would be $2^{23} \times 2^{23}$ or 1 in 70,000,000,000,000. This is actually a gross underestimate because it omits the complicating factors of crossing-over, consanguinity, and many other factors.

I have given an account of this case for several reasons. First, because it illustrates how identical identical twins can be in their development, and second because it is the kind of case that causes many people to make the sort of unjustifiable inferences they do. This type of inference takes the form: if the genes act as powerfully as they do, as in the case illustrated above, then they must

act as powerfully for all other traits, and therefore it is genes with which we have to reckon rather than the environment.

This kind of reasoning, common as it is, is unsound on several counts. It neglects to consider the nature of the environment. It neglects the evidence which shows that there are different degrees of susceptibility to the environment of different developmental processes leading to different traits.

Even in identical twins the expression of genes is influenced, as we have seen, by the environment with which they interact. If the environment has been similar the expression of the genes will be similar; if it has been different the traits "underwritten" by the genes will be different to the extent of the differing environment's influence. The action of the same environmental influences at different times often exerts different effects. The fact that identical twins may often be virtually identical in all respects does not mean that genes alone are responsible, for genes alone are never absolutely responsible for any trait, but what such an identity always means is that the same genotypes of the two individuals have undergone development in a very similar environment. When the environment of identical twins is varied, we observe significant differences developing between them, and this is particularly true of those traits that are most subject to the influence of the environment, namely, psychological traits. An interesting aspect of this is the area of mental aberration or mental disorder.

Mental Disorder in Twins

It has been said by a leading worker in the field of heredity and mental health, Dr. Franz Kallmann, that "the capacities for health and adequate adjustment are fundamental biological properties with the common denominator of hereditary potentiality." This is, of course, true, and it is, of course, also true that adjustment is always conditioned by the interaction of the genotype with the environment. Normal, healthy growth and adjustment can take place only in a normal environment. What is normal, healthy growth and adjustment? It is the process of development without defect, disorder, or disease, the realization of a state of physical and mental well-being. On that subject alone a sizable book could be written, but there is no space for the further development of that definition here.

It is the custom to emphasize the likenesses between identical twins. It is at least as important to draw attention to the differences. Suicide is one of them. It is extremely rare for both twins to commit suicide. In fact, it may be said that the chances are good that if one twin commits suicide the other will not. Suicide is generally the end result of a psychological disturbance of some sort. In most recorded cases the twins were similarly psychologically disturbed; nevertheless only one in the majority of cases committed suicide. Of the total of eighteen pairs of identical twins recorded by Kallmann he found only one set in which both had committed suicide. There is little point to speculating on the reasons for this difference; the fact is that we don't know what they may be in detail, but it seems a not unreasonable conclusion to draw that in general it is to be accounted for on the basis of environmental differences in the experience of the twins.

The reverse of the above findings has been recorded for homosexuals in a series of forty-five pairs of identical twins, in which only one pair were discordant for homosexuality. Kallmann thinks that such findings raise serious questions concerning the psychodynamic theories about the cause of homosexuality and feels that they strengthen the hypothesis of a gene-controlled disarrangement in the balance between male and female maturation (hormonal) tendencies. However, studies on homosexuals do not show such hormonal imbalances (Kinsey), but homosexuals do show a certain similarity in the environment of their early lives. Without entering into a detailed discussion of the matter, it would appear that the vast majority of homosexuals are produced in response to an environment in which one or the other of the parents was markedly inadequate in some way, causing the child to identify itself either with the mother if the father was inadequate or with the father if the mother had failed. Where both parents are inadequate the child, as an adult, may become bisexual (see the works of Henry and Westwood). Hence, without denying the possibility that in some cases homosexuality may have something to do with genes, it is generally agreed among experts that this is a condition which is mainly produced by psychosocial factors during childhood. In spite of the fact that many homosexuals state that they developed these tendencies independently of each other, the possibility must be taken into consideration that they may have been conditioned to do so in early life by similar environmental influences.

A similar interpretation is to be made of many mental disorders which are often attributed to genetic deficiencies, but which upon careful examination quite frequently turn out to be not so much genetic as psychosocial. The fact that a whole family shows a high frequency of mental disorder does not necessarily mean that the family is genetically disposed to develop mental disorder. In many cases it is the environment which is "enough to drive anyone crazy." This is not to say that there are not gene deficiencies underlying many mental disorders. There is very good reason to believe that there are. And these are of two general classes: (1) gene deficiencies related to mental disorder which will express themselves in all known environments, and (2) gene deficiencies related to mental disorder which will express themselves only in certain types of environments. Rather extreme cases conforming to the first class are such disorders as amaurotic idiocy and phenylketonuria, due to rare recessive genes which appear unexpectedly in families in which the condition may never previously have been known to occur. We know of very few other mental disorders which fall into this first class. There is no agreement on what mental disorders may fall into the second class, the reason being that it is extremely difficult to distinguish such disorders—which undoubtedly exist—from those disorders which are more or less purely functionally determined. It would appear that all possible human genotypes are capable of becoming functionally disordered as a consequence of environmental factors, and this applies to physical functions as well as to mental ones. An individual endowed with as "good" a genotype as could be desired can, as a consequence of a disordering environment, become mentally disordered.

When, then, we are asked to view the evidence of mental disorder in twins, we must always be on our guard against the tendency to attribute to genes what may in fact be due to environment.

The principal mental disorders for which a genetic basis has been claimed are schizophrenia, manic-depressive psychosis, and involutional psychosis.

Schizophrenia is a disorder characterized by fundamental disturbances in reality relationships, feeling, intellect, and behavior. It has been described as a fragmentation or rupture of the ego. Manic-depressive psychosis is characterized by marked emotional oscillation from manic to depressive states and vice versa. Involutional psychosis is a mental disorder characterized chiefly by

TABLE IX
EXPECTANCY OF SCHIZOPHRENIA IN CO-TWINS OF SCHIZOPHRENICS

Investigator		Number of Pairs		Rate of Concordance	
		<i>Two-Egg</i>	<i>One-Egg</i>	<i>Two-Egg</i>	<i>One-Egg</i>
Luxenberger	(1930)	60	21	3.3	66.6
Rosanoff	(1934)	101	41	10.0	67.0
Essen-Müller	(1941)	24	7	16.7	71.4
Slater	(1951)	115	41	14.0	76.0
Kallmann	(1952)	685	268	14.5	86.2

depression, and often carries the associated symptoms of insomnia, worry, guilt, anxiety, and delusional ideas.

In Table IX are set out the rate of concordance of schizophrenia in fraternal and identical twins as found by different investigators in Germany, Sweden, England, and America. From this table it will be seen how very much more frequent the concordance (occurring in both twins) of the disorder is in identical than in fraternal twins. The environments are generally as similar for fraternal twins as they are for identical twins, so that if we are to attribute a large part of the role played in producing schizophrenia to the environment, there ought to be many more schizophrenic pairs among the fraternal twins than we actually find. It is estimated that slightly under 1 percent of the general population suffers from one form or another of schizophrenia. Penrose has estimated the frequency of the gene involved as occurring in 1 out of 200 individuals. On the basis of the twin studies it is difficult to resist the conclusion that a significant proportion of these have some sort of genetic susceptibility to the disorder—but always, and until evidence to the contrary is forthcoming, we must remember, under the environmental conditions provocative of the disorder. If some cases of schizophrenia have a genetic basis, it has been suggested that it may be in the form of a recessive gene or genes with variable penetrance and expressivity, depending upon the resistance which has been built up within the individual.

It has been found, for example, that where one identical twin develops schizophrenia and the other does not, the disordered twin is usually weaker physically and of lower weight. Frequently when the general health of the disordered twin is improved and

weight is gained, the schizophrenic symptoms disappear and the individual may resume a normal life.

Manic-depressive psychoses in the general population, occur in about 1 out of every 200 persons. Figures given for the concordant occurrence of the psychosis in identical twins run as high as 90 percent and more. The condition is thought to be due to a dominant gene with varyingly incomplete penetrance.

Involutional psychosis, in the general population, occurs in between 3 to 7 out of every 1,000 persons, and is recorded as occurring concordantly in about 61 percent of identical twins. It occurs with significantly marked frequency among relatives of schizophrenics. It is not considered to be due to a single gene—the genetic mechanism is obscure.

Epilepsy is a neurological disorder which appears to fall into a wide variety of classes with respect to cause. Gene deficiencies are undoubtedly involved in many cases. More than 66 per cent of identical twins have been found to be concordant for the disorder, whereas only 3 per cent of fraternal twins show such a concordance. From such twin studies we are able to deduce that in many cases, if not in all, epilepsy depends on a gene-specific type of vulnerability, in which a number of genes (polygenes) are involved and are most probably carried in the recessive condition. In other words the genes involved must come together from each of the parents in order to express themselves in some of the offspring.

As the reader may have gathered, we know comparatively little concerning the biology of the nervous disorders discussed, and still less of their genetics, but some form of genetic deficiency must be involved in most, if not in all, of them.

Twins have been used in a large variety of studies in order to throw some light on the heredity-environment problem. The studies that we have dealt with here must suffice as typical of the results yielded, inconclusive as they are on many points. There is one area, however, in which twin studies have been made, namely, in behavior disorders, particularly with reference to criminal behavior, that we must consider—and this we may proceed to do in the next chapter.

Crime—Genes or Environment or Both?

A CRIME is an act committed in violation of the law. There are serious students of criminals who have asserted that criminals are born, not made. The late Professor Earnest Hooton of Harvard was one such investigator. His claims have been thoroughly demolished, as have been those of Cesare Lombroso, the Italian criminologist, who initiated criminological studies during the last century, and whose views as to the physical "stigmata" by which criminals might be recognized were widely disseminated throughout the Western world. Ever since Lombroso serious attempts have been made to throw some light on this question, but most of them have suffered from fatal errors both of method and of interpretation which have vitiated their conclusions.

The approach to the problem of the causation of crime through the study of twins is by far the most satisfactory that has thus far been made. Three German, one Dutch, and one American investigator have independently reported on criminality in twins. Their findings are extremely interesting—they are set out in Table X. When both members of a twin pair were found to be similar with respect to the commission of one or more crimes, they were termed "concordant," when dissimilar—that is, when one was found to have committed a crime and the other not—they were termed "discordant." From Table X it will be seen that of 104 pairs of one-egg twins examined, 70 were concordant and 34 were discordant. The concordant were somewhat more

than twice as numerous as the discordant pairs. On the other hand, the two-egg twins showed a discordance somewhat more than twice as great as the concordance shown in this group of 112 pairs. The proportions are exactly reversed between the one-egg and the two-egg twins.

These are interesting figures, but what do they mean? It has been held by several authorities that they mean that genetic factors play a large part in the causation of criminal behavior. This is the opinion of the five investigators whose twin studies we are here considering. The truth, however, is that such studies do not prove any connection whatever between genetic factors and criminal behavior. One-egg twins are likely to have the same companions and to be otherwise closely associated in social activities; they are therefore more likely to encounter together such social influences as may lead them to criminal activities. All our knowledge of twins tells us that two-egg twins do not experience environments as closely similar as those of one-egg twins. Hence, it is quite reasonable to account for the greater concordance of criminal behavior among one-egg twins on the basis of the greater similarity of their environmental experience as compared with the two-egg twins.

Unfortunately, the tendency has been to underplay the significance of the environmental factor in the interpretation of the findings in such twin studies of criminality. Once more the danger is underscored of illegitimately assuming that a higher frequency of concordance of any behavioral trait in one-egg twins as compared with two-egg twins indicates the greater likelihood of the operation of purely or partly genetic factors in the one-egg twins than in the two-egg twins. Where the environment has been highly similar this is a distinctly dangerous inference to make, even if in some cases it turned out to be true. On the evidence that is available to us most of the time, however, it is an erroneous inference, for what is attributed to genes may, in fact, be largely or wholly due to environment.

The proportion of two-egg twins who are both affected and the proportion of one-egg twins where only one is affected are virtually identical, being 33 percent for two-egg concordance, and 32.7 percent for one-egg discordance. Discordant one-egg twins ought not to be so frequent and discordant two-egg twins ought to be more frequent, according to the genetic theory.

TABLE X
CRIMINAL BEHAVIOR OF TWINS

Investigator		One-Egg Twins		Two-Egg Twins	
		Con- cordant	Dis- cordant	Con- cordant	Dis- cordant
Lange	(1929)	10	3	2	15
Legras	(1932)	4	0	0	5
Kranz	(1936)	20	12	23	20
Stumpfl	(1936)	11	7	7	12
Rosanoff	(1934)	25	12	5	23
Total		70	34	37	75
Percent		67.3	32.7	33.0	67.0

The actual findings, however, reveal that one third of the one-egg pair of twins investigated were discordant. Why did not the genetic factor for crime declare itself in the other member of this one third of single-egg twins? Perhaps it did, but he had the luck not to get caught. If, however, the answer is that an environmental factor was operative in these cases, a factor which was absent in the case of the criminal sibling—or if you like, an environmental factor was operative in the case of the criminal twin and was absent in the case of the law-abiding one—then the theory of the genetic causation of criminal behavior collapses beyond repair, for it then becomes clear that it was the absence of some environmental factors, or the presence of others, which constituted the indispensable condition in the causation of the criminal behavior. We surely cannot assume that the environmental conditions were the same for both twins, with their identical genotypes, when one becomes a criminal and the other does not. If the difference is not to be found in their genes the only other place it can be looked for is in their environment.

There almost certainly exist differences among individuals of a generalized genetic kind which, *under certain kinds of environmental conditions*, may make some individuals more ready than others to commit acts designated by society as criminal, but such environmental conditions are not like triggers which set off or release inborn tendencies to commit criminal acts. There are no such things as inborn tendencies to speed in a nonspeeding zone,

to fail to return borrowed books, to commit petty larceny, robbery, felonious assault, arson, rape, or murder, or any other kind of social offense. But what do exist are pressures of the environment which, together with specific psychosocial histories, cause the individual to commit such offenses *regardless of the nature of his genotype*. As we know, the genotype can never be entirely disregarded, but for many practical purposes it certainly can be disregarded in the sense that its causal contribution to a particular social effect may have been negligible or none at all. If, for example, all human genotypes were identical but social environments varied as they do today, there would still be marked differences in crime rates, and it is greatly to be doubted whether they would appreciably differ from those of the present time.

It is not "criminal genes" that make criminals, but in most cases "criminal social conditions," regardless of whether such "criminal" conditions are created by society or the family. The conditions that drive men to crime are the effects of their accumulated environmental experiences, whether those environmental experiences are accumulated in a criminal or delinquent environment where moral and ethical standards are virtually nonexistent, or whether they are accumulated in homes where there is no love but only hostility, frustration, or indifference. Delinquents are not born; they are made by a delinquent society, which makes delinquent parents, and who make the kind of delinquent environments with which we are familiar in the Western world.

Why is it that delinquency and crime are virtually unknown among Chinese immigrants and their children living in the United States? How does it happen that drunkenness is a condition that occurs so rarely among Jews? Is it because these groups have few or no genes disposing them toward such behavior? The answer is positively in the negative. The infrequency of criminal behavior among American Chinese and of drunkenness among American Jews is no more genetically influenced than is the tendency of Chinese immigrants to become laundrymen and of Jews to attend synagogues. These are all forms of behavior that can be shown to be determined by cultural and not by biological factors. The Chinese are brought up in an environment in which gentleness, kindness, and honesty are traditionally strongly emphasized. When they emigrate to America it is usually with the support of a rela-

tive who is already engaged in laundry work. Jews do not get drunk as often as their neighbors do because in the home drinking is largely regarded as a ritual act and is usually limited to wine. Because of the high negative sanctions that are enjoined against such a condition, drunkards are held in contempt. When Chinese and Jews become thoroughly Americanized and lose the ancient virtues of their own cultures, some of them become as proficient in the vices of their adopted culture as any of their fellow Americans.

What shall we say of the millions of murders committed on helpless individuals by the Germans during World War II? Shall we say that in the pre-Hitler environment the genetically determined impulses of the Germans to murder were kept under control, but that with the advent of Hitler and his sanction of such murderous behavior, the environment was changed and the murderous impulses were released? This could be persuasively argued, but surely it is nearer the truth to say that in every country there are millions of frustrated individuals with violent impulses suppressed inside them, produced by the frustrative conditions of their lives, who would welcome a socially sanctioned opportunity to expend their hostilities in some way. It is no more true to say that it is genes that make nations murderous than it is true to say that it is genes that make men so.

Human beings are not born with genes for being "good" or "bad." Nor are they born like blank tablets upon which one can write whatever one desires, for the organism is characterized by developmental limits which are largely determined by its genes. However, within those genetic limits what the organism as a behaving individual becomes will largely, if not entirely, be determined by its environmental experience. The discrepancy between what we are and what we were capable of becoming constitutes for many of us the real tragedy of life.

It is amusing to record the reply of the great hereditarian among criminologists, Cesare Lombroso, when he was confronted with his own student's investigation, that of Enrico Ferri, who found that 63 percent of soldiers showed Lombroso's so-called "stigmata of degeneration" (lobeless ears, small ears, receding chins, and the like). Lombroso attempted to explain this startling discovery away with the statement that when the "stigmata are found in honest men and women, we may be dealing with criminal

natures who have not yet committed the overt act because the circumstances in which they have lived protected them against temptation."

So we see that in the end what Lombroso stated was that individuals exhibiting stigmata will be prone to commit crimes under certain environmental conditions. Since almost all individuals exhibit one or another of these so-called stigmata, we may unreservedly agree. Under certain environmental conditions all of us are capable of committing crimes, and most of us have. But what Lombroso meant, and always insisted upon, was that in almost all cases it was not the unfavorable environment which led to the commission of a crime, but the biological predisposition to commit it, which could be foretold by the presence of stigmata. The stigmata were taken by Lombroso to be marks of biological inferiority, "atavisms" * (Latin *atavus*, ancestor; hence, process of reversion to an ancestral state), proofs of the reversion to more primitive forms of biological organization which was reflected in primitive levels of response or behavior. This criminal behavior was inseparably connected with biological inferiority; the biological inferiority was held to be the cause of criminal behavior.

As we have already said, attached ear lobes, low foreheads, receding chins, malformed ears, crooked noses, the so-called stigmata of Lombroso, are traits that are widely distributed among the populations of every land, and it is now known that there is no genetic relationship between such physical traits and behavior. Malformed features may, with the assistance of an unsympathetic environment, cause the individual to develop maladjusted behavior, but, again, such responses have nothing whatever to do with genes.

Hooton, unlike Lombroso, did not set out with any preconceived notion as to what criminal stigmata might be: he allowed the sample of 4,212 white prisoners of Old American stock who were the subject of his investigation to tell him what these marks were. Hooton took the marks of biological inferiority to be any of the characters which were distinctive of the criminal aggregate when compared with the civilian sample. "Thus," Hooton writes in his book *Crime and the Man* (1939), "if we find felons to mani-

*There are no such things as "atavisms" or "throwbacks." See Montagu, "The Concept of Atavism," *Science*, vol. 87, 1938, pp. 462-463.

fest physical differences from civilians, we are justified in adjudging as undesirable biological characters those which are associated in the organism with antisocial behavior. . . . It is the organic complex which must be estimated inferior or superior on the basis of the type of behavior emanating from such a combination of parts functioning as a unit. . . .”

“Whatever the crime may be,” Hooton concludes, “it ordinarily arises from a deteriorated organism . . . You may say that this is tantamount to a declaration that the primary cause of crime is biological inferiority—and that is exactly what I mean.”

When Hooton’s “marks of inferiority” are examined, it is found that they consist mostly of measurements of the body, indices expressing one dimension as a percentage of the other, and measures of form. For his marks of inferiority Lombroso took characters which were for the most part apelike. If we classify Hooton’s marks of inferiority by standards which are generally accepted to be marks of primitiveness, advancement, or as neutral, then Hooton’s results when thus analyzed are by no means surprising. By such standards we find that of Hooton’s combined anthropometric, indicial, and body-form characters only 4 percent are primitive, 15.8 percent are neutral, and 49.5 percent are *advanced* characters, the last appearing more frequently than among the noncriminal population.

By biological standards, therefore, we see that Hooton’s findings actually make his criminal series a considerably more advanced group biologically than his noncriminal series of 313 native-born white civilians.

Dr. W. H. Sheldon has in recent years claimed to have found a relationship between body type and delinquent behavior. In a study of 200 delinquent youths in Boston he found that the 16 criminals in the series belonged to a rather broad-chested muscular type called the endomorphic-mesomorphic type. Sheldon does not suggest that this type of body build “predisposes toward criminality, but,” he says, “it might mean that to make a go of being a criminal requires a certain amount of guts that is usually found only” in this body type. But that, of course, is precisely the point. There is a certain amount of social selection at work in criminal as well as in many other types of social and antisocial activities. Clearly, the robust, big-chested, tough-looking male has a great advantage over the roly-poly fatty or the long, linear,

stringlike type in any activities involving the boldness of muscularity and the necessity of violence. But Sheldon is inclined to attribute this selection more to what he calls "guts" than to "occupational" requirements. In this opinion he stands with Hooton, with whom he identifies himself. As Eleanor and Sheldon Glueck point out, body type may be significantly related to delinquency, but can by no means be regarded as a cause of it. Boys with a long, linear body type (*ectomorphs*) may be teased a great deal, so may roly-polies (*endomorphs*), while broad-chested muscular types (*mesomorphs*) may be selected by certain environments for delinquent roles by other routes.

We may conclude, then, that all biologically biased attempts and other more dispassionate attempts to prove a relationship between genes or "heredity" or "constitution" and criminal behavior have failed. Crime appears to be the result of a complex of factors and there is very good reason to believe it has but little, if any, relation to genetic or constitutional factors. The significant relationship is to social factors. Everyone agrees that there is a high correlation between poverty and crime, although, of course, criminals are by no means drawn exclusively from the poorer classes. And poverty by itself is rarely a cause of crime, but within the group of conditions which constitute the cause of crime in any one instance it is of frequent occurrence. No kind of genotype of which we have any knowledge is associated with criminal behavior. The evidence indicates that all individuals, whatever their genotype, as a result of exposure to certain social conditions, can be caused to function in a criminal manner. There is every reason to believe that with changes of the social environment in the right direction criminal behavior could be reduced to the vanishing point.

Genes and Constitution

“IT’S CONSTITUTIONAL.” That statement generally carries the weight and finality of a Supreme Court judgment. If it’s constitutional there is nothing one can do about it except by constitutional amendment. Perhaps we can achieve a change in our understanding of what we mean by the “constitution” of human beings more easily than we can in that noble body of laws, the Constitution of the United States. Somehow the word “constitution” as applied to human beings has come to mean the bodily and functional expression of the biological endowment, the genes, of the individual. This is the biologicistic fallacy once again at work. The constitution of the individual may quite properly be defined in terms of his bodily make-up and functions, but it is quite another thing to attribute that make-up and those functions to the exclusive determination of genes. “Constitution” is defined as the physical make-up of the body, including the mode and performance of its functions, the activity of its metabolic processes, the manner and degree of its reactions to stimuli, and its power of resistance to the attack of pathogenic organisms. All the criteria or terms of this description of constitution are determined neither by genes nor by environment, but by the *interaction* of genes and environment. The magnitude of the role played by the one or the other varies with the kinds of conditions which make up the elements of constitution. In some conditions the genes are almost exclusively responsible; in others the environment seems to play the major determining role. But the process of interaction between the two is always involved.

Normally when we use the word "constitution" it is in application to the postnatal organism, and in most cases we tend to use the term with respect to adults, but whether we use the term in connection with children or adults, we use it in a kind of final sense, meaning, "This is what this individual is" or "This condition is the result of the biological structure of this individual," and in either case there is nothing one can do about it because it is the individual's biological fate. In no case is this pessimistic conclusion sound, not even in those cases in which it is possible to predict with complete certainty that they will end in death. If there is one thing that can be predicted with complete certainty, it is that we shall all die, and most of us will die before we reach the age of eighty. But even in those cases in which we can predict that the individual will not live to attain five years, as in the case of amaurotic family idiocy, in which the infant usually dies before he reaches his third year, it is now possible to prolong the life of such an individual appreciably. In some cases it may be debatable whether this is at all desirable. But in any event even in the worst of cases we do not have to sit back in resigned hopelessness. For if we are aware that constitution is not something fixed and final but is something about which, even in the worst of cases, we *can* do something, however little it may be in the present state of our knowledge, there is the hope that we may someday be able to do much more. And by citing the facts, this is what I want to make quite clear in the present chapter.

It should be clearly understood that there is little that is terminal about constitution, for constitution is a *process* rather than a fixed, final, and unchangeable entity. It is important to understand that constitution is not a biological *given*, a structural system predestined by its genotype to function in a predetermined manner. As, I hope, has been sufficiently emphasized in these pages by this time, the manner in which all genotypes function is determined by the interaction of the genotype with the environment in which it undergoes development. What, so to speak, the genotype—that complex of genetic potentialities with which the organism is endowed—asks is: "What kind of responses are going to be made to my chemically accelerating and developmental overtures, my tentative advances? How will I impress? How will I be impressed? For the outcome of all this will be my constitution."

The point that must be emphasized here is that every genotype

is a unique physicochemical system comprising particular kinds of potentialities having definite limits. These limits vary from individual to individual, so that were the genotype to be exposed to identical environmental conditions its interactive expression would nevertheless continue to vary from individual to individual. But in point of fact the environmental conditions never are the same for two individuals, not excluding one-egg twins. This fact renders it necessary for us to recognize that constitution is not merely constituted by the genotype, but by the genotype as modified by the environment in which it has developed, as the result of the dynamic interaction between the two. As the sum total of the structural, functional, and psychological characters of the organism, constitution is in large measure the integral expression of the genetic potentialities influenced in varying degrees by internal and external environmental factors.

Let us now proceed to examine the constitutional factor in relation to the conditions with which it is most frequently discussed, namely, disease. It is often implied that when a disease is stated to be constitutional that that is the long and the short of it, it will always be with the individual, and there is very little or nothing that can be done to alter the situation. The diseases discussed in what follows are all cited in order to show that this view is unsound and requires modification in the light of the facts.

Diabetes

Diabetes has already been cited as a constitutional disease about which a great deal can be done (see page 75). True diabetes (diabetes mellitus) is caused in most cases by the failure of the pancreas to secrete a sufficient amount of its hormone, insulin. The consequent failure in the proper conversion and utilization of sugar results in its excessive accumulation in the blood, with seriously damaging results to the organism. There is no means of curing genetically conditioned diabetes at the present time, but the sufferer from the disease can be maintained on insulin for many years as an efficient and to all intents and purposes a comparatively healthy human being. In other cases in which the disease is caused by an excessive secretion of hormones from the pituitary or adrenal glands, removal of one or other of these glands may alleviate the

condition. One out of every four diabetics comes from a family in which one or more relatives are also diabetic. In the many cases that are of genetic origin the condition is due to a pair of recessive genes. In other cases it is suspected that the condition may be produced by emotional disorders combined with overeating and similar disordering states. Thus in the genetic cases environmental means may alleviate the condition; in the other types of cases the stress of environment may bring on the condition, which by the appropriate means may be either alleviated or altogether cured.

By making simple glucose tolerance tests it is possible to diagnose the prediabetic state and prevent the development of full diabetes in these individuals.

Tuberculosis

Tuberculosis is a disease produced by the tubercle bacillus. But susceptibility to the disease presents marked individual differences and even familial differences. Since the disease is highly infectious, it is to be expected that several members of a family will show it, and this is, indeed, the case, but there are many families in which only one member develops the disease, while the others, though they may have been infected with the tubercle bacillus, show not the slightest evidence of the disease. The fact is that millions of human beings have been infected with the tubercle bacillus but have not become diseased. Indeed, only a small proportion of those who do become infected become diseased. These facts strongly suggest that there exist genetically influential differences in resistance to tuberculosis. The breeding of different strains of rabbits has actually been accomplished which show marked differences in resistance to tuberculosis. This takes the form of differences in the virulence with which the disease attacks these different strains of rabbits. Kallmann and Reisner found that while only 25.6 two-egg twins were concordant for the disease (the same rate as for ordinary brothers and sisters), 87.3 one-egg twins showed concordance. The probability of a genetic factor connected with susceptibility to tuberculosis is therefore high, and it would appear likely that many genes are involved. However, it is clear from these same twin studies that in the discordant cases where there is a differ-

ence in the health of the twins it is invariably the weaker twin who is affected. So the resistance is not entirely genetically determined. And the truth is that the best protection against tuberculosis seems to be the development of a healthy body in a healthy environment. Resistance to tuberculosis, then, can be built up by building up the health of the individual. Essentially this means the provision of good conditions for prenatal and postnatal development and the maintenance of those conditions throughout the life of the individual. This also implies a general improvement in the conditions of the environment. As a result of the general betterment of the health of the average individual and the improvement of the environment during the last sixty years, there has been a spectacular decline in the tuberculosis disease and death rates. In 1900 there was an annual death rate from tuberculosis of 194 persons per 100,000 of population. By 1950 this was down to only 20 persons per 100,000. This means that in the year 1950 instead of there being 315,000 deaths at the 1900 rate, there were only 35,000 deaths, a saving in that year alone of 280,000 lives. This saving was undoubtedly due to an improvement in the constitution of many individuals as well as to a general improvement in environmental conditions and improved methods of detection and treatment. Vaccination with B.C.G. (*Bacillus Calmette-Guérin*) affords protection against infection with TB. No matter what the constitutional predisposition of the individual may be to tuberculosis, if he contracts the disease its course can be modified by the proper environmental procedures, especially by treatment with antibiotics such as streptomycin.

The principles involved in the modification of the course of tuberculosis, its prevention, and cure are applicable to all diseases, whether they are only partly conditioned or wholly determined by genetic factors.

Being born with a genetically determined disorder does not condemn, as it always did in the past, the individual to the consequences which inevitably followed. We are now able to control the expression of some genetically determined disorders, and had we possessed the knowledge of these matters fifty years ago that we possess today, the whole course of world history might have been altered. I have in mind the famous case of hemophilia, or "bleeder's disease," as it worked out its tragic consequences in Russia.

Hemophilia

Hemophilia or "bleeder's disease" is a disorder characterized by a defect in the clotting power of the blood, and as a consequence of this, intractable and profuse bleeding following the slightest scratch, cut, or bruise may result in death. In former years very few hemophiliacs managed to survive into adulthood. But since there are varying degrees of severity with which the disorder manifests itself, there have always been a number of adults of quite advanced age who were hemophiliacs.

Hemophilia is caused by a sudden change in the chemical structure of a normal gene situated on an X chromosome. Because the effects of such a defective mutant gene are always linked with the sex chromosome, and because it is carried in the recessive condition, the hemophilia gene is recognized as a sex-linked recessive. It has been calculated that the mutation from the normal "big *H*" to the hemophilic gene, "little *h*," occurs in every generation in 1 out of every 50,000 persons. This is a relatively high rate of mutation and means that in the English-speaking world at the present moment there are probably over 10,000 persons in whom the hemophilia gene has come into being quite spontaneously. To see how simple the genetic mechanism of hemophilia transmission is, let us take an actual case.

The normal gene for blood-clotting undergoes mutation in a man. This occurs in his X chromosome, for his Y chromosome has no corresponding gene. This man marries a normal woman with two healthy X chromosomes. They have five sons and five daughters. Since the male transmits his X chromosome to his daughters only, they will all become carriers of the defective gene, but because they have inherited a healthy gene for clotting in the X chromosome they have received from the mother, the disorder will not express itself in them. That is why it is exceedingly rare for a female to be afflicted with this condition. What would be necessary for a female to suffer from hemophilia would be the inheritance of a defective gene on the mother's X chromosome and a defective gene on the X chromosome contributed by the father. Several such cases have been recorded. Since the male with the defective gene for blood-clotting does *not* transmit his X chromosome to his male offspring, the sons of a hemophilic

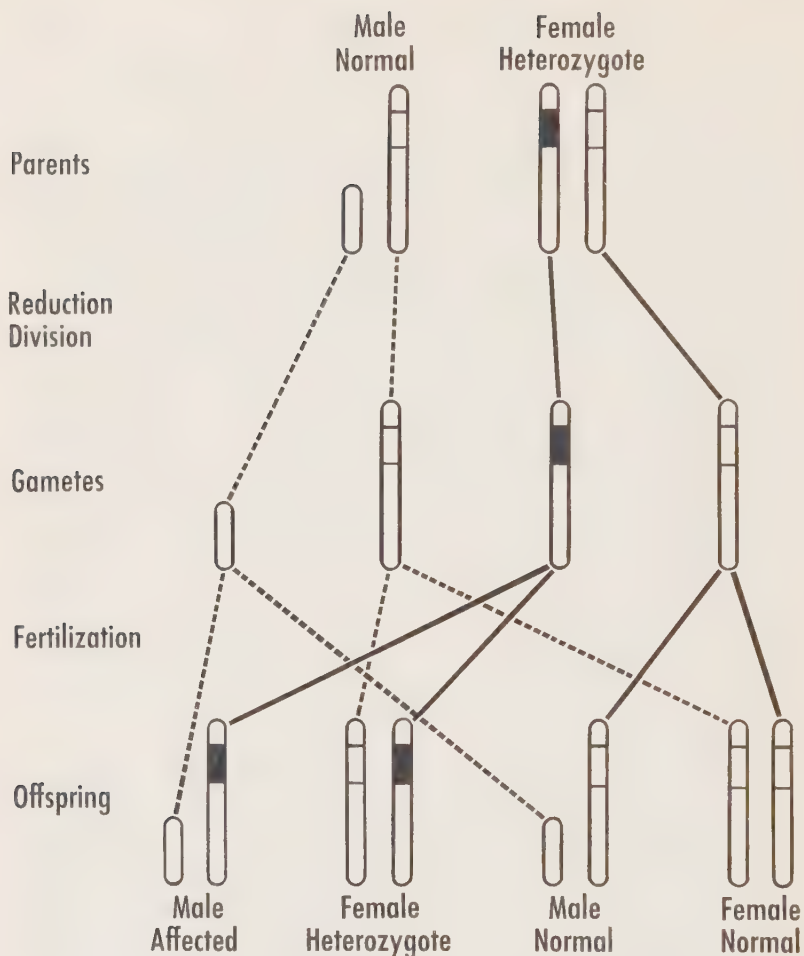


FIG. 18. The Transmission of the Hemophilia Gene.

father never suffer from the disorder except in the rare event that the father married a carrier. His daughters, marrying normal men, will transmit the defective X chromosome to about half their sons and to half their daughters, but the sons will be hemophilic while the daughters receiving the defective chromosome will not, for their "good" X chromosome protects them from the "bad" one,

whereas the male has only a Y chromosome which contains nothing that can afford him protection.

Hemophilia is one of the most tragic of disorders. The most famous family line affected by it is that of Queen Victoria (1819-1901). Victoria was the daughter of the Duke of Kent, the fourth son of George III. It is believed that the mutation from the normal gene for blood-clotting to the abnormal gene occurred in the person of Queen Victoria's father, that he transmitted his defective X chromosome to her, with the consequences set out in figure 19. One of Victoria's four sons, Leopold, Duke of Albany, was a hemophiliac. He married and transmitted the defective gene to his descendants. Two of Victoria's daughters transmitted the disorder to the royal families of Russia and Spain. The heirs to both thrones were hemophiliacs, and there can be little doubt that this fact played a considerable role in leading to the revolutions which occurred in both countries. The czarevitch was the answer to his mother's devoted prayers. She had given birth to four girls when finally an heir to the throne was born. The tragic blow that befell her when the czarina learned that her son was a hemophiliac is one from which she never recovered. This caused her to resort to soothsayers, quacks, and mystics of every kind, and this is what led her to fall under the influence of the scheming monk Rasputin, who claimed to work remarkable cures and whose influence at court created that atmosphere of intrigue and duplicity which alienated millions of Russians and helped prepare the way for the Revolution. Neither the dethronement of the czar or of Alfonso XIII of Spain was the direct result of the fact that their sons and heirs were hemophiliacs, but there can be little question that the knowledge that the successors to the throne were invalids played an important role in bringing about the fall of these monarchies. Today the discovery that there are different varieties of hemophilia and hemophiloid conditions and the discovery of some of the factors that enter into the causation of these conditions have made it possible to alleviate, at least, the severity of these conditions. The expectation is that further intensive research will yield much more successful modes of treatment. In January, 1959, a new coagulant for treating hemophilia became available. This antihemophilic globulin, which consists of a white powder obtained from the blood of pigs, is nearly twenty times as potent in its antihemophilic properties as are human blood trans-

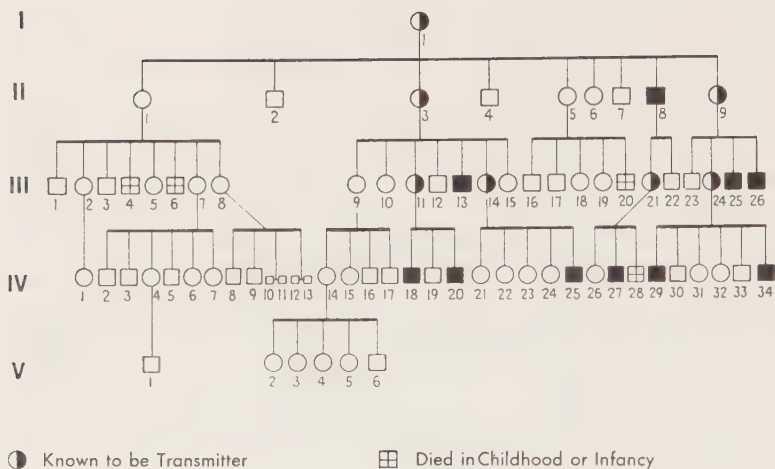


FIG. 19. Hemophilia in Queen Victoria's Descendants.

I. (1) Victoria, 1819–1901, Queen of England.

II. (1) Victoria, 1840–1901, × Frederick III, Emperor of Germany.
 (2) Edward VII, 1841–1910, King of England. (3) Alice, 1843–1878, × Prince Louis of Hesse. (4) Alfred, 1844–1900, Duke of Edinburgh. (5) Helena, 1846–1923, × Prince Christian of Schleswig-Holstein. (6) Louise, 1848–1939, × Duke of Argyll. (7) Arthur, 1850–1942, Duke of Connaught. (8) Leopold, 1853–1884, × Princess Helena of Waldeck. (9) Beatrice, 1857–1944, × Prince Henry of Battenberg.

III. (1) William II, 1859–1941, Emperor of Germany. (2) Charlotte, 1860–1919, × Duke of Saxe-Meiningen. (3) Henry, 1862–1929, Prince of Prussia. (4) Sigismund, 1864–1866. (5) Frederica, 1866–1929. (6) Waldemar, 1869–1879. (7) Sophie, 1870–1932, × Constantine, King of Greece. (8) Margaret, 1872–1954, × Frederick, Duke of Hesse-Cassel. (9) Victoria, 1863–1950, × Prince Louis of Battenberg (Marquess of Milford Haven).

fusions. So far the treatment can be used only once to save a bleeder's life, but it is expected that soon a purified preparation will become available that can be used whenever it is needed.

Still other examples of constitutional conditions may be cited which, by altering the constitution of the individual, may save his life.

(10) Elizabeth, 1864–1918, × Grand Duke Sergius of Russia. (11) Irene, 1866–1953, × Prince Henry of Prussia [see III (3)]. (12) Ernest, 1868–1937, Grand Duke of Hesse. (13) Frederick William, 1870–1873. (14) Alexandra, 1872–1918, × Nicholas II, Czar of Russia. (15) Mary Victoria, 1874–1878. (16) Christian Victor, 1867–1900. (17) Albert, 1869–1931. (18) Victoria, 1870–1948. (19) Marie Louise, 1872–1956. (20) Harold, 1876–1876. (21) Alice, 1883–, × Earl of Athlone. (22) Charles Edward, 1884–1954, Duke of Albany. (23) Alexander, 1886–, Marquess of Carisbrooke. (24) Victoria Eugénie, 1887–, × Alfonso XIII of Spain. (25) Leopold, 1889–1922, Lord Mountbatten. (26) Maurice, 1891–1914, Prince of Battenberg.

IV. (1) Feodora Maria, 1879–1898, × Henry XXX of Reuss. (2) George II, 1890–1947, King of Greece. (3) Alexander, 1893–1920, King of Greece. (4) Helena, 1896–, × Carol II of Romania. (5) Paul, 1901–, King of Greece. (6) Erene, 1904–. (7) Catherine, 1913–. (8) Frederick William, 1893–1916, Prince of Hesse. (9) Maximilian, 1894–1914. (10) Philipp, 1896–. (11) Wolfgang, 1896–. (12) Richard, 1901–. (13) Christoph, 1901–. (14) Alice, 1885–, × Prince Andrew of Greece. (15) Louisa, 1889–, × King Gustav VI Adolf of Sweden. (16) George, 1892–1938, Marquess of Milford Haven. (17) Louis, Lord Mountbatten, 1900–. (18) Waldemar, 1889–1945, Prince of Prussia. (19) Sigismund, 1896–1927. (20) Henry, 1900–1904. (21) Olga, 1895–1918, Grand Duchess. (22) Tatiana, 1897–1918, Grand Duchess. (23) Marie, 1899–1918, Grand Duchess. (24) Anastasia, 1901–1918, Grand Duchess. (25) Alexis, 1904–1918, Czarevitch. (26) May, 1906–, × Sir Henry Abel-Smith. (27) Rupert, 1907–1928. (28) Maurice, 1910–1910. (29) Alfonso, 1907–1939, Prince of Asturias, later Count of Covadonga. (30) Jaime, 1908–. (31) Beatrice, 1909–. (32) Maria, 1911–. (33) Juan, 1913–. (34) Gonzalo, 1914–1934.

V. (1) Michael, 1921–, King of Romania. (2) Margarita, 1905–, × Godfrey of Hohenlohe-Langenburg. (3) Theodora, 1906–, × Margrave of Baden. (4) Cecilie, 1911–1937, × Grand Duke of Hesse. (5) Sophie, 1914–, × Christopher of Hesse. (6) Philip, 1921–, Duke of Edinburgh.

Hereditary Hemolytic Icterus

Hereditary hemolytic icterus is a very serious form of jaundice combined with anemia. It is inherited as a dominant autosomal gene and is due to the enlargement of the spleen, which keeps too many red blood cells out of circulation and gradually destroys

them. The result is jaundice and anemia and often very rapid death. It is not only possible to save such persons by a timely removal of the spleen, but it is possible to prevent the development of the disease in those individuals whom it would certainly kill, by the simple device of examining the offspring and immediate relatives for any signs of the precursors of the disease. Examination of their blood for changes in number, size, shape, variety of cells, and certain simple reactions makes it possible to determine whether the disease is likely to declare itself in any of the examined individuals. Removal of their spleens, which in no way seems to have any untoward effects, will save them from much suffering. In this manner many lives have already been saved that were formerly doomed. Many tragedies have thus been averted, and we have cases on record where several members of a family have willingly undergone the removal of their spleens and thus saved their lives, while other members of the same family have refused to undergo the operation and have died as a result.

Incipient cases of hypertension, pernicious anemia, certain types of cancer, and many other genetically based disorders have also been diagnosed and prevented from developing in a similar way.

From the cases cited it will be observed that not only can the effects of a constitutional disorder be alleviated, but in many instances it can also be prevented from developing. Hence, the simple conclusion can be drawn that one's constitution is not unalterable or unmodifiable, that it is not the equivalent of implacable fate, but that it is in many respects quite amenable to the changes induced by environmental means.

Sex

THE TERM “sex” (Latin *sexus*) is probably derived from the Latin verb *secare*, meaning to cut or divide. Sexual species are divided into male and female. It is by means of the union of the sexes that new gene combinations are brought into being in an ever-varying but constant variety. By this means the species is reproduced and maintained from generation to generation. The great variety of genotypes thus produced endows the species with a high degree of adaptability, so that under gradual or sudden changes in the environment there are generally likely to be genotypes that will be able to respond to the challenges of the changed environment. The genotypes unable to make the necessary responses are unlikely to leave a sizable progeny behind them.

On the other hand, in organisms that usually reproduce asexually, with no division into male and female sexes, like the one-celled amoeba or the slipper-shaped, one-celled paramecium, variability is reduced to a minimum. Among such organisms reproduction usually occurs by simple fission or budding-off, an exact duplicate thus being formed which varies ever so slightly from the maternal or sister organism. Different sets of genes are usually not brought together and recombined in the offspring, so that it will be understood why evolutionary change is likely to be arrested until sexually reproducing species make their appearance. Sexual reproduction is the great conduit through which most biological change is established within the species. By whatever agencies changes are produced in organisms, it is only

through the reproductive process that they can be distributed within the population, and in some groups may even be made to cross over from one species and even one genus to another.

In all sexual species it is the female who harbors the eggs, eggs with which, as in the human species, she is already endowed—to the number of about half a million—at birth. The male, on the other hand, is endowed with sperm-forming organs, the testes, which gradually develop the capacity to manufacture new sperm, of which he manufactures many billions in the course of a lifetime. The human female sheds her eggs on the average about every twenty-eight days from about (on the average) the age of sixteen to about the age of fifty years, whereas the male normally continues to manufacture spermatozoa till he is beyond sixty, and is not infrequently capable of producing fertilization in a young female at eighty years and beyond. Records of the offspring of such unions show that they are perfectly normal in every way.

While the eggs age in the aging female, apparently the organs producing the sperm cells in the male preserve their integrity till an advanced age. There is undoubtedly some adaptive advantage in this arrangement from the evolutionary standpoint. The explanation that suggests itself is that since there are always fewer males in any population than females, there will always be an excess of females. During the early evolution of man, it would have been of advantage to the group if an older man whose wife had ceased to be fertile were able to take an additional younger woman or two as members of his family. We still find this custom (polygyny) very widely distributed among nonliterate peoples. From the standpoint of the survival of the group with a small population, the custom has very obvious advantages.

The female has a much shorter reproductive life than the male, both (1) during her twenty-eight-day cycle (ovulation occurs from the fourteenth to the seventeenth day, counting from the first day of menstruation, with the ovum fertilizable for about two days at most during that period after it has entered the fallopian tube), and (2) in the total duration of her capacity to reproduce, which lasts about thirty-five years. In addition, the human female takes, on the average, $266\frac{1}{2}$ days from conception to delivery to gestate a child, whereas it takes the male only a few minutes to initiate the process that eventually leads to the fertilization of an egg. With these facts the reader may begin to perceive that the main-

tenance of the species is rather more dependent upon the female than it is upon the male. In order for the species to be maintained, the female must be impregnated, and this can be achieved within a very narrow range of time. Once impregnated, she must be preserved for at least $266\frac{1}{2}$ days if the species is to be perpetuated. The male who produced the pregnancy, however, could die immediately after conjugation, even before fertilization had occurred, and the continuity of the species would be quite unaffected.

For all these reasons (and a good many more) the female is the biologically more valuable part of the species capital—and this is true for similar reasons throughout the whole realm of animated nature. She has to be constitutionally stronger, genetically more resistant than the male, and, indeed, in every species characterized by sexual reproduction thus far investigated, the female has proved so. It is unlikely that any exceptions will be found to this fundamental rule, for it reflects a basic difference in the biological structure of the sexes, the very difference that determines sex itself.

As I have already explained, until April, 1959, it was believed that females owe their sex to the fact that they have acquired one X chromosome from their mother and another X chromosome from their father; whereas males owe their sex to the fact that they have acquired one X chromosome from their mother and a Y chromosome from the father. In 1958 Drs. J. H. Tjio and T. T. Puck measured the X and Y chromosomes and found the X chromosome to be three times larger than the Y chromosome. Each X chromosome contains a full complement of genes, whereas the Y chromosome probably does not. Until 1956 it was thought that it did contain genes for over-hairy ears, horny, scalelike, or barklike skin (*ichthyosis hystrix gravior*), a nonpainful nodular affection of the hands and feet, and webbing between the second and third toes. But the first case is based on a single pedigree, the second has been shown to be unsound, and the two last conditions occur in both males and females, which renders it something less than certain that Y chromosome inheritance is involved in any of the very few cases reported. Although we do not know precisely what genes the Y chromosome contains, we can be reasonably certain that it contains *some* genes, but the existence of these remains to be demonstrated. In fact, every condition of which we have positive and verifiable knowledge which is linked in heredity with a

sex chromosome is *always linked with the X chromosome*, and these conditions are known as *sex-linked* conditions. Hemophilia, which we have already described, is an example. We shall shortly return to a discussion of sex-linkage. What we have to emphasize here is that the female possesses two complete X chromosomes and the male possesses only one. It is this difference which endows the female with that biological superiority which, as we shall see, enables her to resist the assaults and insults of the environment so much more effectively than the male.

In April, 1959, publication of two papers, one by W. L. Russell, Liane Brauch Russell, and Josephine S. Gower, and the other by W. J. Welshons and Liane Brauch Russell, all of the Oak Ridge National Laboratory, Tennessee, showed that the Y chromosome is vitally important in sex determination in mammals. This is in contrast to *Drosophila* where sex is determined by the balance between X chromosomes and autosomes, with the Y playing a neutral role. By ingenious breeding and cytological studies involving certain exceptional mice, it was found that these animals, which were females of almost normal productivity, were of chromosome constitution XO. That is, they carried only one X and therefore had only 39 chromosomes instead of the normal 40. This is clearly different from *Drosophila* where XO animals are males. The results in the mouse show that the Y chromosome is the bearer of the male-determining factors. They also indicate that all female-determining factors are situated on the autosomes, or, as seems more likely, that the X chromosome carries at least some female-determining factors.

If the X chromosome carries some female determiners then in those cases in which the organism has acquired an extra sex chromosome (as a result of non-disjunction at either mitosis or meiosis) and is of constitution XXY, we should expect either an intersexual or possibly even a female. There is some suggestive evidence of this in the literature, and in January, 1959, Jacobs and Strong of the University of Edinburgh reported such an apparently XXY condition in an intersexual young man of a nuclear sex chromatin type indicating a genetic female.

If we ask ourselves how it comes about that the female possesses two X chromosomes and the male only one, we may conjecture that the possession of a complete set of X chromosomes endows the female with a greater capacity for survival

than the male. By biologically strengthening the female, the higher probability of the survival of the species is brought about. This interpretation of the facts the reader will be able to make for himself as he follows the evidence in the succeeding pages.

Differential Survival Rates of the Sexes

Since 50 percent of spermatozoa carry an X chromosome in their heads, and 50 percent carry a Y chromosome, it would seem that half the eggs fertilized should be females and half should be males. But this is not the case. Y-bearing sperms fertilize between 120 and 150 eggs for every 100 eggs fertilized by X-bearing sperms. The same ratio of difference has been found also in other mammals. The reason for this difference in fertilization is unknown. It has been suggested that Y-bearing sperms swim faster than X-bearing sperms. This is almost certainly not the explanation, for in birds, in which the chromosomal contents of the germ cells are reversed (that is, eggs are of two kinds, X-bearing, and Y-bearing, whereas male sperms contain only X-bearing chromosomes), males conceived still outnumber the females conceived. On the average at each successful coitus the male ejaculates over 200,000,000 spermatozoa. Only one of these, if it reaches an egg, will produce fertilization. Whatever the physical reasons may be which result in more eggs being fertilized by Y-bearing spermatozoa the evolutionary "reason" would appear to be that *since the male is the constitutionally weaker organism he must be conceived in greater numbers than the female if a relatively harmonious numerical balance is to be achieved between the sexes during the reproductive life of the female*. We have already observed that the greater duration of reproductive capacity in the male compensates somewhat, insofar as the survival of the species is concerned, for his average lesser overall durability.

At birth for every 100 females that are born there are about 105 males born. This means that out of the 120 or 150 that have been conceived, between 15 and 45 males have died *in utero*. But this is true only if we take the view that every female conceived survives to birth, and this is not the case. Some females also die *in utero*. We do not know how many. Evidently fewer female conceptions fail to come viably to term than male ones. At every stage of prenatal and postnatal development the mortality rate

TABLE XI
MORTALITY RATES FROM EACH CAUSE BY SEX
WHITE POPULATION OF CONTINENTAL UNITED STATES FOR THE YEAR 1957
RATES PER 100,000

Cause of Death	Male	Female
Tuberculosis of respiratory system	9.5	3.1
Tuberculosis, other forms	0.5	0.3
Syphilis and its sequelae	2.5	0.9
Virus diseases	1.6	1.5
Malignant neoplasm of buccal cavity and pharynx	5.6	1.5
Malignant neoplasm of digestive organs and peritoneum	59.1	48.3
Malignant neoplasm of respiratory system	36.1	6.3
Malignant neoplasm of breast and genito-urinary organs	28.4	58.2
Malignant neoplasm of other and unspecified sites	17.6	15.0
Neoplasms of lymphatic and hematopoietic tissues	17.2	11.9
Benign neoplasm	1.0	1.6
Neoplasm of unspecified nature	1.7	1.3
Allergic disorders	5.5	2.4
Thyroid diseases	0.2	1.0
Diabetes mellitus	12.9	19.0
Other endocrine gland diseases	0.5	0.5
Avitaminoses and other metabolic diseases	0.8	0.7
Pernicious anemia	0.4	0.5
Hemophilia	0.1	0.0
Psychoses	0.7	0.6
Psychoneurotic disorders	0.0	0.1
Disorders of character, behavior, and intelligence	2.1	0.5
Cerebral hemorrhage	63.6	67.9
Meningitis	1.1	0.8
Multiple sclerosis	0.8	1.0
Other diseases of central nervous system	5.4	4.2
Epilepsy	1.3	0.9
Motor neurone disease and muscular atrophy	0.8	0.5
Diseases of ear and mastoid process	0.3	0.2
Rheumatic fever	0.5	0.4
Arteriosclerosis and degenerative heart disease	292.2	242.1
Other diseases of heart	14.4	9.3
Aortic aneurysm, nonsyphilitic	6.4	2.2
Peripheral vascular disease	0.3	0.1
Thrombo-angiitis obliterans	0.2	0.1

Based on *Vital Statistics—Special Reports, National Summaries*, vol. 50, No. 2, February 4, 1959, U. S. Department of Health, Education, and Welfare, Public Health Service, National Office of Vital Statistics, Washington, D.C.

TABLE XI
MORTALITY RATES FROM EACH CAUSE BY SEX
WHITE POPULATION OF CONTINENTAL UNITED STATES FOR THE YEAR 1957
RATES PER 10,000

Cause of Death	Male	Female
Arterial embolism and thrombosis	0.3	0.2
Varicose veins, lower extremities	0.1	0.2
Hemorrhoids	0.1	0.0
Varicose veins, other sites	0.3	0.1
Influenza	4.2	3.6
Lobar pneumonia	8.8	5.2
Bronchopneumonia	18.0	13.5
Primary atypical pneumonia	2.9	2.2
Pneumonia, other and unspecified	4.0	2.9
Bronchitis	2.8	1.3
Bronchiectasis	1.9	0.9
Stomach ulcer	4.5	1.6
Duodenal ulcer	5.2	1.4
Gastrojejunal ulcer	0.3	0.1
Gastritis and duodenitis	0.2	0.1
Appendicitis	1.5	0.7
Abdominal hernia	2.3	1.9
Gastro-enteritis and colitis	2.1	2.0
Chronic enteritis and ulcerative colitis	1.7	1.7
Peritonitis	0.4	0.4
Cirrhosis of liver	15.8	7.5
Cholelithiasis	1.7	2.4
Cholecystitis	0.9	1.2
Other diseases of gall bladder and biliary tracts	0.5	0.8
Diseases of pancreas	1.6	1.1
Chronic nephritis	6.9	5.7
Hydronephrosis	0.2	0.1
Cystitis	0.2	0.1
Rheumatoid arthritis	0.4	0.7
Osteitis deformans	0.1	0.0
Myasthenia gravis	0.1	0.2
Inborn defect of muscle	0.5	0.2
Curvature of spine	0.1	0.1
Congenital malformations	13.8	11.5
Intracranial and spinal injury at birth	2.7	1.5
Other birth injury	4.9	3.4
Postnatal asphyxia and atelectasis	11.1	7.7
Pneumonia of newborn	2.0	1.3
Other diseases peculiar to infancy	17.7	12.9

is higher for the male than the female. At every age during postnatal life more males are dying than females.

Fetal deaths are 50 percent higher in males than in females. Within the first month following birth the male death rate exceeds that of the female by 40 percent, and among prematures by 50 percent. Within the first year of life male mortality exceeds that of the female by 33 percent. For ages five to nine male mortality exceeds that of the female by 44 percent, from ten to fourteen by 70 percent, and from fifteen to nineteen by 145 percent. The disparity in the rates rises steadily till twenty-one, when the death rate of the male exceeds that of the female by 130 percent, and then grows increasingly less, though it never at any time reaches equality with that of the female.

The statement is often made that men die earlier on the average than women because they work harder than women. This statement is a good example of the kind of mythology to which many of us subscribe even in the face of the facts to the contrary.

Newborn boys do not work harder than newborn girls; yet they die more frequently than girl babies. One-year-old boys do not work harder than one-year-old girls, but the boys die more frequently than the girls. And so one can go on for every age, with the difference in mortality being in favor of the female.

It is found that the married man lives longer on the average than the bachelor, and that the married woman lives longer than the spinster, so, if anything, the state of marriage helps both sexes to greater longevity. Men and women engaged in the same kind of work, and men and women who don't work, still show mortality rates that are in favor of the female. A recent study, published in 1957, of the longevity of Catholic Sisters and Brothers who for many years had been living the same kinds of lives showed the same sort of disparity in their mortality rates as the rest of the population. The data were obtained on nearly 30,000 Sisters and more than 10,000 Brothers. The investigator, Father Francis C. Madigan, working at the University of North Carolina, found that the expectation of life at the age of forty-five was 34 years more in the Sisters and only 28 years in the Brothers, the difference favoring the Sisters being $5\frac{1}{2}$ years.

At the present time the average male child born in the United States may expect to attain an age of about 67.4 years, whereas the average female child may expect to attain an age of more than 73.6 years. Table VII on pages 120-121 gives the expectation

of life at birth for a number of countries. It seems most unlikely that we shall be able to close the gap that exists between male and female longevity in the near future, but as we learn more about the nature of the hereditary factors involved, it is highly probable that we shall not only be able to lengthen the life of the male and reduce this gap, but also lessen the prenatal and postnatal mortality rates that exist for both sexes. When we have succeeded in doing even part of this, new social problems will be facing us as a result of the unusual increase of males, unless, of course, we foresee the nature of those problems and do something to control the proportions of the sexes born so that they are harmoniously balanced. This brings us to the matter of the artificial determination of sex. We shall deal with this later on in this chapter, but now we must discuss the mechanisms involved in producing the sexual differences in the rates of functional capacity, sickness, and death.

As I have already explained in connection with the manner of inheritance of hemophilia, the possession of a double number of X chromosomes protects the female against the development of this deficiency disorder even though she has inherited the defective gene which is the cause of the condition in the male. It is this protection afforded her by the extra X chromosome which confers a basic biologically determined natural superiority upon the female as compared with the male who possesses only one X chromosome. Genes for traits or characters which are associated in inheritance with sex are of three kinds: (1) *sex-linked*, (2) *sex-influenced*, and (3) *sex-limited*.

Sex-Linked Genes

When a trait is conditioned by a gene lying in the X chromosome it is said to be *sex-linked*. What we mean by sex-linkage is that the X chromosome carries genes for characteristics other than those which merely determine sex. This means that such characteristics will be linked with sex in heredity. It does *not* mean that such characteristics are linked to a particular sex but that such characteristics will follow the distribution of the X chromosome in both sexes. However, because the male carries only one X chromosome any defective genes present in that chromosome are likely to express themselves considerably more frequently in him than in the female.

Let us consider the X chromosome. It contains genes that are lacking in the Y chromosome. These genes will always express themselves in the male offspring because there is nothing present in the Y chromosome which can possibly affect their expression. The effects of such genes appear in the female only if the gene is present in both X chromosomes. The genes passed on in this manner are said to be wholly sex-linked. More than thirty traits are known to be due to sex-linked genes. These traits are shown in Table XIII. We have already seen how sex-linkage works in the case of hemophilia. Let us deal with another sex-linked condition, namely, color blindness. There are many different kinds of color blindness. At one extreme are the persons who cannot distinguish any colors. At the other extreme are those persons who have some slight defect in their ability to distinguish red from green. Red-green blindness is the commonest of all inherited defects which show a sex-linkage. Among whites 2 men out of every 25 suffer from some lack of ability to recognize red and green. Less than 1 woman out of 200 suffers from a similar defect.

One way in which color blindness can be inherited is shown in figure 20. The dark X is an X chromosome that carries the gene for color blindness. If a man inherits such a gene, he will be color-blind. There is no corresponding gene in his Y chromosome that can counteract the effect of the defective gene in his X chromosome. If such a man marries a woman with two normal X chromosomes, all the children will receive a normal X chromosome from their mother. The daughters will also receive an X chromosome from their father which carries the gene for color blindness. They will usually have normal vision. The gene for color blindness is recessive; that is to say, it is a gene which will not normally express itself in the presence of the normal gene, which is dominant. The recessive gene has no effect on the phenotype unless it is carried in the homozygous state or is carried on an X chromosome in the male. Thus, a person receiving both a defective gene and a normal gene will be normal for the trait; a person receiving only the defective gene will exhibit defective red-green vision. The gene for normal vision is said to dominate the defective gene for red-green vision in the female. In the male if the gene is normal there will be normal vision; if it is defective there will be defective vision. Since the male never transmits his X chromosome to his sons, but the female transmits one of her

TABLE XII
SEXUAL DIFFERENCES IN SUSCEPTIBILITY TO DISEASE

MALES		FEMALES	
Diseases	Preponderance	Diseases	Preponderance
Acute pancreatitis	Large majority	Acromegaly	More often
Addison's disease	More often	Arthritis deformans	4.4-1
Amebic dysentery	15-1	Carcinoma of genitalia	3-1
Alcoholism	6-1	Carcinoma of gall bladder	10-1
Angina pectoris	5-1	Cataract	More often
Arteriosclerosis	2.5-1	Chlorosis (anemia)	100%
Bronchial asthma	More often	Chorea	3-1
Cancer, buccal cavity	2-1	Chronic mitral endocarditis	2-1
Cancer, G.U. tract	3-1	Combined sclerosis	More often
Cancer, head of pancreas	4.5-1	Diphtheria	Slight
Cancer, respiratory tract	8-1	Gall stones	4-1
Cancer, skin	3-1	Goiter, exophthalmic	6 or 8-1
Cerebral hemorrhage	Greatly	Hemorrhoids	Consid.
C.S. meningitis	Slight	Hyperthyroidism	10-1
Childhood schizophrenia	3-1	Influenza	2-1
Chronic glomerular nephritis	2-1	Migraine	6-1
Cirrhosis of liver	3-1	Multiple sclerosis	More often
Coronary insufficiency	30-1	Myxedema	6-1
Coronary sclerosis	25-1	Obesity	Consid.
Duodenal ulcer	7-1	Osteomalacia	9-1
Erb's dystrophy	More often	Pellagra	Slight
Gastric ulcer	6-1	Purpura haemorrhagica	4 or 5-1
Gout	49-1	Raynaud's disease	1.5-1
Heart disease	2-1	Rheumatoid arthritis	2-1
Hemophilia	100%	Rheumatic fever	Consid.
Hernia	4-1	Tonsilitis	Slight
Hodgkin's disease	2-1	Varicose veins	Consid.
Hysteria	2-1	Whooping cough	2-1
Korsakoff's psychosis	2-1		
Leukemia	2-1		
Mental deficiencies	2-1		
Muscular dystrophy, Ps.h.	Almost exclusively		
Myocardial degeneration	2-1		
Myocardial infarction	7-1		
Paralysis agitans	Greatly		
Pericarditis	2-1		
Pigmentary cirrhosis	20-1		
Pleurisy	3-1		
Pneumonia	3-1		
Poliomyelitis	Slight		
Progr. muscular paralysis	More often		
Pseudohermaphroditism	10-1		
Sciatica	Greatly		
Scurvy	Greatly		
Syngomyelia	2.3-1		
Tabes	10-1		
Thromboangiitis obliterans	96-1		

Source: Montagu, *Introduction to Physical Anthropology*.

TABLE XIII
SOME HUMAN TRAITS REPORTED AS DEPENDENT UPON SEX-LINKED GENES

Albinism of the eyes	Megalocornea
Alopecia congenita	Microcornea
Anhidrotic ectodermal dysplasia	Microphthalmos
Coloboma iridis	Mitral stenosis
Color blindness of the red-green type	Myopia
Day blindness	Night blindness
Defective hair follicles	Nomadism
Defective tooth enamel	Nystagmus
Distichiasis (double eyelashes)	Optic atrophy
Epidermal cysts	Peroneal atrophy
Glaucoma of the juvenile type	Pseudohypertrophic muscular dystrophy
Hemophilia	Retinal detachment
Ichthyosis	Thromboasthenia
Keratosis	White occipital lock of hair

Adapted from Snyder, L. H., "The Mutant Gene in Man."

two X chromosomes to all her children, the sons of a color-blind mother, no matter whether her husband has normal or defective red-green vision, will all be color-blind, but the daughters will all have normal color vision if the father is normal in this respect. But these daughters all become carriers of the recessive gene for color blindness, and marrying a normal man they will transmit the defective gene to about half the sons, and these sons will be color-blind because they have no masking gene in their Y chromosome that compensates for the defective gene. Half the daughters will receive the defective gene from their mother. They will not be color-blind because the normal X chromosome inherited from their father will be dominant over the gene for color blindness. These daughters in turn will be able to transmit color blindness to half their sons. Color blindness in a daughter can only be produced if a color-blind man marries a color-blind woman or a woman who was carrying the defective gene in the recessive state.

Color blindness is an important problem in a society in which, to cite but one example, the ability to distinguish between red and green traffic signals may mean the difference between life and death. Many color-blind persons have learned to distinguish between the different traffic lights not by their color but by their

standard position in the light. Even so, color blindness does present a small but definite social problem. What should be done with the color-blind? One solution would be to prevent them from having children. Another, and obviously far more sensible, would be to make color filters available to them so that they could easily detect essential color differences.

In the male sex-linked genes can be represented on only one chromosome, the X chromosome. Sex-linked genes can be represented on both chromosomes in females. From these facts and the fact that almost all sex-linked mutant genes are recessive, it is possible to give specific advice to families known to be carrying such genes. If the men in such a family do not exhibit the trait, then clearly the gene for that particular expression of the

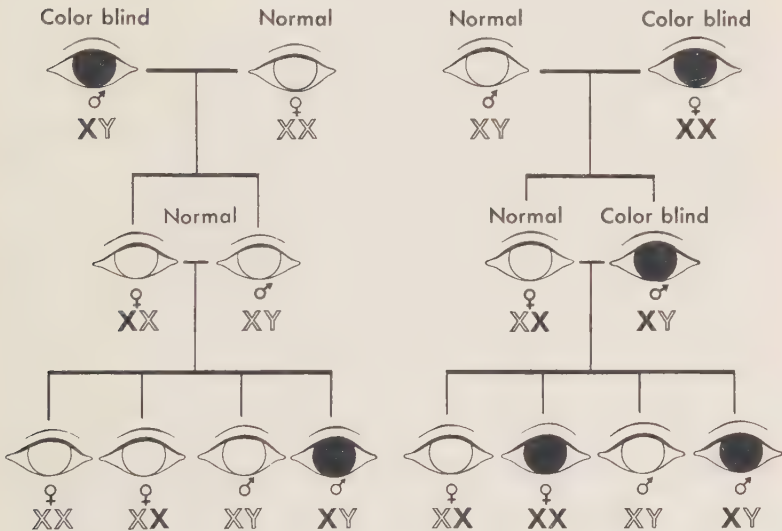


FIG. 20. The Inheritance of Color Blindness. 1. In the mating of a color-blind man with a normal woman, since the defective gene is sex-linked it is transmitted through the daughters of this mating, and appears in half of their sons. 2. In the mating of a color-blind woman with a normal man, the defect is transmitted to all the sons but to none of the daughters who are, however, carriers. Such a carrier is mated to a color-blind man in this diagram, in the second parental generation, and half the grandsons and half the granddaughters are color-blind. (After Dunn.)

trait is not present on his X chromosome, and he cannot therefore transmit the gene for the condition to any of his offspring. He may safely have children. An affected father can transmit his defective X chromosome to his daughters, who will *all* be carriers. Since the sons receive only his unaffected Y chromosome from him *none* of them will be affected. The sons may safely marry. When the carrier daughters marry a normal man, as they are most likely to do, they will transmit the defective X chromosome to half their daughters and to half their sons. The daughters will be unaffected but half the sons will be.

There are some conditions which appear to be due to *incompletely sex-linked genes*. The manner in which these conditions behave in inheritance leads to the conclusion that the human Y chromosome must possess some genes. If incompletely sex-linked genes are present in both chromosomes of both sexes, they will behave like autosomal genes in inheritance, with this difference: About half the families in which the fathers carry the gene will contain more affected sons and unaffected daughters than would be expected on the basis of autosomal inheritance, while the other half will contain more affected daughters and unaffected sons than would be expected on the basis of autosomal inheritance. The reason for this is that such genes can cross over from the X to the Y chromosome, and vice versa in the male, and thus will be differentially transmitted as to sex. If the male receives the defective gene from his mother he transmits it to a majority of his daughters and a minority of his sons.

About ten or so traits have been attributed to incompletely sex-linked genes. These are: total color blindness (as opposed to red-green blindness), xeroderma pigmentosum (a childhood skin disease characterized by numerous pigmented spots, lesions, and a glossy-white thinning of the skin, often terminating fatally), Oguchi's disease (a type of blindness), spastic paraplegia (a neuromuscular defect), the recessive form of epidermolysis bullosa (malignant skin blisters), the dominant form of retinitis pigmentosa, hereditary hemorrhagic diathesis (a blood abnormality), and a type of cerebral sclerosis (a mental defect).

Sex-Influenced Traits

Sex-influenced traits are conditioned by genes carried in the autosomes, and hence are inherited equally by both sexes and

transmitted equally by both sexes. Sex, however, controls the dominance. The gene which is dominant in one sex is recessive or intermediate in the other, and vice versa. Several sex-influenced traits have been described in man, such as one form of white forelock, absence of the upper lateral incisor teeth, simple ichthyosis (scaling of skin), and Heberden's nodes (enlargement of terminal joints of fingers). Though baldness has customarily been attributed to sex influence, it is in fact an example of sex limitation and not of sex influence. We shall deal with baldness under sex-limited traits.

An example of a sex-influenced trait is provided by a form of white forelock described by Holmes and Schofield in 1917, which appeared in four generations in a family of 32 individuals in 14 males but was not present in 3 males nor was it present in 15 females. It could be transmitted either through the males or the females and was dominant in males and recessive in females, and hence was sex influenced.

Sex-Limited Traits

When certain traits are expressed in one sex but not in the other they are said to be *sex limited*. Sex-limited characters are conditioned by genes which are carried either in the autosomes or in the sex chromosomes. There are, therefore, many different types of sex-limited heredity. The expression of sex-limited traits depends largely upon the presence or absence of one or more sex hormones, or to put it more accurately, the amount of such hormones present within the organism. Complete sex limitation, that is, complete development of the trait in one sex and complete absence of it in the other sex, is not frequent. Examples of complete sex limitation are milk production and menstruation in females but not in males. Another example of such complete sex limitation is the appearance of coarse hairs on the external ear of white men during the process of aging, and the absence of such hairs in women. I am not aware of any evidence indicating that the administration of male hormone to women at any age will induce the growth of such hairs in their ears.

Another example, but not quite so complete, is the presence of a developed beard and mustache in the male and their absence in the female. It has been shown that women have exactly the same number of hairs on the face as men, but the hairs sim-

ply do not develop and grow as they do in men. However, under the proper endocrine stimulation with male hormones women are capable of growing fairly respectable beards and mustachios.

A familiar example of sex limitation is baldness. That baldness is not due to a sex-linked gene is clear from the fact that bald fathers, depending upon whether they carry two or only one baldness gene, transmit their baldness to all or only half their sons, and it will be remembered that since sex-linked characters are conditioned by those carried in the X chromosomes, a man cannot transmit X-linked genes to his sons, but only to his daughters in whom they remain recessive. Furthermore, hereditary baldness in the female is extremely rare. In the male baldness, in varying degrees (see fig. 22), occurs in more than 40 percent of those over the age of 34 years. Baldness can show up in males when neither parent is bald, when one or both parents contribute a baldness gene to their sons. A bald father may have some sons who become bald and some who never become bald or all the sons may become bald in varying degrees. Daughters, however, will experience thinning or partial baldness, but complete hereditary baldness of the masculine type in the female is of ex-

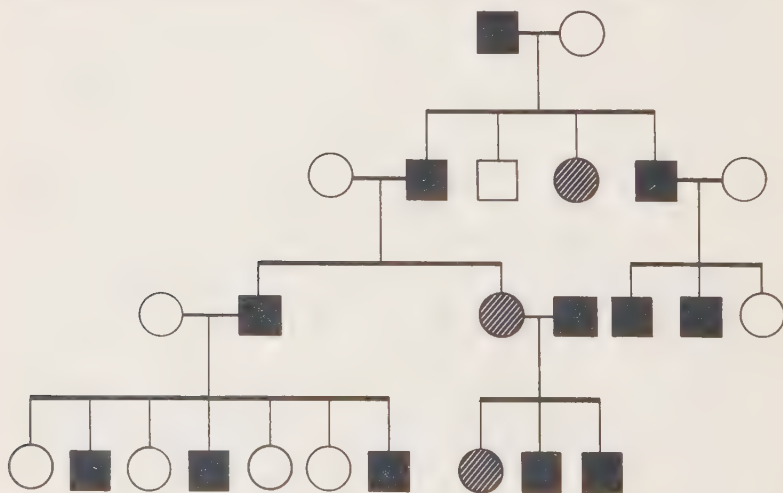


FIG. 21. A Family Pedigree of Balding.

Solid symbols indicate balding in males. Cross-hatched symbols indicate females showing thinning of hair.



FIG. 22. Patterns of Balding. Types I and II are non-bald. Types III to VII represent a graded progression of common pattern baldness. (Modified after Hamilton.)

cessive rarity. When baldness of the masculine type affects a female it is usually due to disease or disorder. As Obermayer and others have shown, baldness in varying degrees may be produced in both sexes by psychogenic factors, hence the statistics on baldness in women patients in mental hospitals which have been used in some books to interpret the genetic mechanism of baldness have only served to confuse matters.

In some cases baldness is due to disease of the hair follicles and in others to hormonal disorders, but these are the exceptional cases. In most cases baldness is due to the action not of genes that have been acquired from the parents, but to the action of hormonal factors upon these genes. The genes for baldness de-

rived from father and mother are identical, but their action, their expression, differs very markedly in each sex. Obviously the fact that the same genes express themselves differently in male and female is another way of saying that the same gene acts differently in each sex owing to some difference in the constitution of the sexes. The nature of this constitutional difference is suggested when, as not infrequently happens, a woman develops a tumor of the adrenal gland or within the ovary. Often such a tumor will produce abnormal stimulation of certain cells causing them to secrete large amounts of male sex hormones (androgens). These androgens produce a masculinizing effect upon the woman causing, among other things, reversal of secondary sexual characteristics, including those of larynx, voice, and mental attitudes, and the development of a mustache and beard. Some women do not, under such conditions, develop thinning of the head hair, which suggests that they probably do not carry any baldness genes. When the tumor is surgically removed, the masculinized woman rapidly returns to her female traits, though some residua such as the masculine voice and Adam's apple may remain, but the tendency is for the mustache and beard unsustained by male sex hormone to return to normal, and this is also likely to be the case where head hair has thinned. Hence, it is clear that the presence of male hormones has something to do with baldness. Investigation of this possible association in the male has, indeed, revealed that there is a very significant association between these two phenomena.

Experiment has shown that no amount of male sex hormone will produce baldness in males who have not inherited the necessary genes. It has long been known that men who have been castrated in youth, eunuchs, never develop baldness. On the other hand, some males, who for one reason or another have suffered a more or less extreme reduction in the quantity of these hormones circulating in their bodies, when male sex hormones are administered to them undergo a loss of hair. In the actual cases to which I am referring it was noticeable that the men came from families with a tendency to hereditary baldness. It was also found that in those men who came from families with no hereditary tendency to baldness that the administration of male sex hormones had no effect whatever upon the growth of their head hair—baldness could not be produced in them.

HOW BALDNESS IS INHERITED

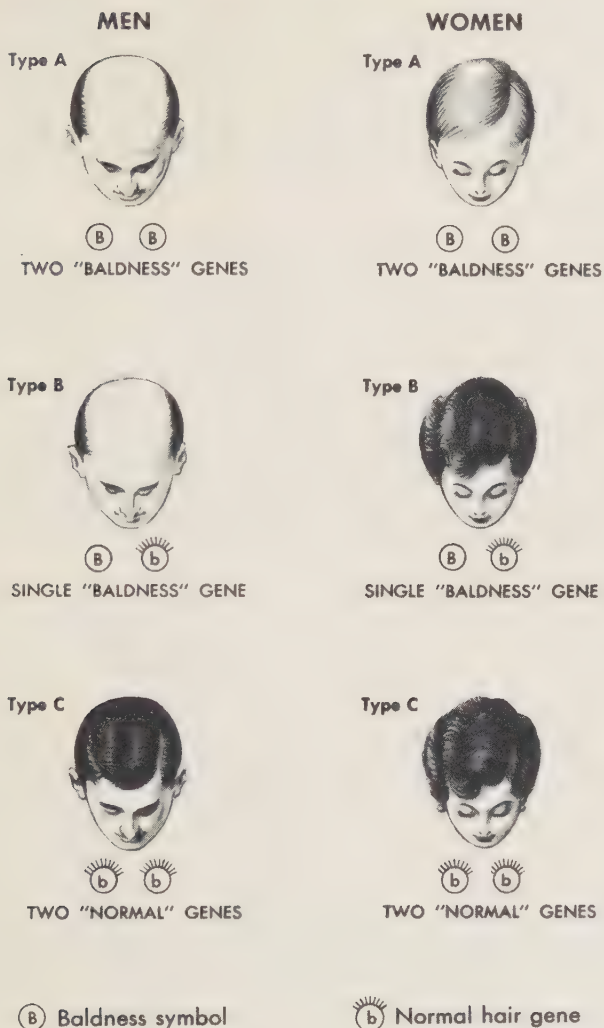


FIG. 23. Baldness is a sex-limited trait due to dominant gene which is fully expressed in the male, but limited in its expression in the female. (Modified after Scheinfeld, *The New You and Heredity*.)

The sex-limited effect of baldness is, therefore, clear. The baldness gene must be present if baldness is to occur, but baldness will occur very much more frequently in the male than in the female—even in the attenuated form which it may take in her—principally because the male's sex hormone enables the gene to express itself more completely. Or put in another way, the male's sex hormone is more devastating upon the hair follicle in the presence of the baldness gene than is the female sex hormone in relation to the baldness gene.

The expressive or dominance relations of the genes for scalp hair growth seem, then, to be sex controlled or limited. It is not that baldness is a dominant trait in the male and recessive in the female. If this were so then in the case of two recessive baldness genes in the female baldness would result, but we know that it does not in fact do so. The genes involved, we have already said, are the same in each sex, it is simply that their expression is differentially limited by the factor of sex. The hereditary mechanism is quite simple. If big *B* is the gene for baldness, and little *b* the gene for nonbaldness then the following genotypes and phenotypes appear:

<i>Genotype</i>	<i>Males</i>	<i>Females</i>
<i>BB</i>	Bald	Thinning of hair or partial baldness
<i>Bb</i>	Bald	Nonbald
<i>bb</i>	Nonbald	Nonbald

In other words a Baldness gene derived from one parent and a Baldness gene derived from the other parent will express themselves in double dose in the male, but will be limited in their expression in the female only to the extent of thinning or partial baldness. Where the Baldness gene has been derived from one parent and the *b*, nonbaldness gene, from the other parent, the Baldness gene will express itself in baldness in the male, but the Baldness gene associated with the nonbald *b* allele in the female will remain unexpressed, and the female will therefore be nonbald. A nonbald gene *b* derived from one parent and a nonbald gene *b* derived from the other parent will always yield nonbaldness in whatever sex they come together. Figure 21 shows an actual pedigree of baldness which demonstrates the effects of sex limitation.

Since almost all cases of baldness are genetically determined, and there is at present no known means of influencing this genetically determined sex-limited trait, all claims made through advertisement or otherwise promising to alleviate or "cure" the condition must be regarded as fraudulent. This does not mean that some time in the future a way may not be found for controlling baldness. It probably will. But at the present time no such way exists.

The difference between sex influence and sex limitation is that the same trait when it is *sex influenced* (the genes being carried in the autosomes) can be equally frequently expressed in both sexes, but that in one sex the trait will appear to be transmitted as a dominant, while in the other it will act as if its expression were due to a recessive. Whereas in *sex-limited inheritance* (genes carried on both autosomes and sex chromosomes) the trait is fully expressed in one sex alone. It is not the genes that condition the expression of the trait but the sex of the individual.

Gout, for example, is an *incompletely sex-limited* trait because while the gene is dominant and is carried on the autosomes, it is expressed in about 95 percent of males and only in 5 percent of females. Gout does not occur equally frequently in both sexes, but is largely limited to males, occurring in females not only in a much smaller proportion of cases but in a less exacerbating form. The female constitution exercises a strikingly modifying effect not only upon the expression of certain genes, but even upon the damage that certain infective organisms will work upon her as compared with the male. For example, syphilis follows so benign a course in the female as compared with the male that it has been said that it manifests itself in the female "almost as if she were of another species." This is true of many other conditions.

There are disorders which are inherited as recessives and which should therefore be equally expressed in male and female which are, however, more frequently expressed in one sex than in the other. Such conditions more frequently expressed in the male than in the female are albinism (sometimes dominant), alkaptonuria (black urine disorder), retinitis pigmentosa (progressive inflammation of the retina), and amaurotic idiocy.

Similarly, some conditions which are due to recessive genes are expressed more frequently in the female, such as diabetes (some-

times dominant), manic-depressive insanity, Huntington's chorea, Sydenham's chorea, and Niemann-Pick's disease.

The secondary sex characters, such as mustache and beard, body-fat distribution, form of body, breast development, texture of hair, distribution and growth of body hair, and also many differences in response of the nervous system, depend upon sex-limited factors. The degree of secondary sexual development in the sexes is obviously differently limited by the hormones of the glands of internal secretion (endocrine glands)—the factors of sex are in themselves genetically limited—but this limitation is to some extent under the control of the hormones. This is clearly seen even in prenatal development in the case of the free martin.

A *free martin* is a sterile unsexed female with a tendency toward the development of male characteristics. Similar cases occur in human beings, but not quite in the same manner in which they sometimes occur in cattle when twin calves of opposite sexes are born. In such cases there is a connection between the blood vessels on the outer (chorionic) of the two membranes enveloping the fetuses, and since the male hormones develop earlier than those of the female they get through to the female fetus, inhibiting the normal development of the female organs and serving to masculinize them so that the ovaries tend to form testicular tissue and are incapable of producing ova. In the human species it is the action of freely circulating excessive amounts of androgens which produce masculinization of the female embryo, usually by the sixth week of uterine development.

Modes of Inheritance

It is important to note that the modes of inheritance of the same trait may differ in different families. In one family a trait may be due to a sex-linked dominant gene, in another family the same trait may be due to an autosomal recessive gene, and in still another family to an autosomal dominant gene. Usually this means that the chain of events leading to phenotype A can be switched at different steps to give B, the steps corresponding to different genes that may be anywhere in the chromosomes.

Mutant genes and linkage between such genes occur in autosomes, but their study is considerably more difficult than sex-linkage. Several such linkages have, however, been described.

Examples are the linkages between ear flare and finger length, finger length and eye color, ear size and ability to taste a substance known as phenylthiocarbamide (or P.T.C. for short), hair whorl and cross-eyes, skin color and hair color, eye color and tongue curling, hair shade and hair color, ability to taste mercapto-benzoselenazol (M.B.S.) and ear size, ability to taste M.B.S. and tongue curling, sickle-shaped red blood corpuscles and the MN blood type, Lutheran blood group and the secretor gene, and several others.

A gene that affects two or more parts or characters of the body which have no obvious relationship is termed *pleiotropic* (Greek *pleion*, more; *trope*, a turning). Most genes are pleiotropic. In man this pleiotropism with respect to the pigmentary system, for example, is apparently exhibited in hair, eye, and skin color, dark-skinned people usually having dark hair and eyes, fair-haired people usually having light-colored skins and eyes. One seldom sees black-haired people with blue eyes, although this combination occurs among the Irish, thus indicating that separate genes are at work in influencing the expression of these two traits.

Penetrance, Expressivity, and Viability of Genes

Under the conditions in which genes develop they show varying degrees of activity. They are influenced, as we have seen, by internal and external environmental conditions, and one non-allelic gene may act upon another to inhibit its action (*epistasis*), or may itself be inhibited by a nonallelic gene (*hypostasis*).

Penetrance. When a gene regularly produces the same effect in all the individuals exhibiting it, it is said to have *complete penetrance*. When the effect is not produced in some individuals, even though they carry the gene in either homozygous or heterozygous state, the gene is said to have *reduced penetrance*. Penetrance relates therefore to the *either-or* state of the gene—either it is expressed in the form of a definite condition or it is not. Dominant genes with low penetrance may be mistaken for recessives. The blood groups are an example of genes that show complete penetrance. Every individual inheriting a gene for a particular blood-group trait develops that trait. On the other hand it has been shown that in such a disease as diabetes insipidus (the rarer form of diabetes, not to be confused with the common form, diabetes

mellitus) is probably due to an incompletely dominant gene which manifests itself in about 10 percent of those carrying it.

Expressivity. When the manifestations of a trait vary from individual to individual, the gene is said to have variable expressivity. When the manifestation of a trait is constant, the gene is said to have constant expressivity. The dominant gene for allergy shows variable expressivity and may take such forms as asthma, eczema, hay fever, angioneurotic edema (sudden appearance of urticarial, or hivelike, swellings on face or upper extremities).

Viability. Genes carried in the homozygous state which shorten the life of the individual are known as lethal genes. Lethal genes are incompatible with life at various stages during the development of the individual. This means that such genes may exercise their effect at fertilization and at almost any time thereafter. Most of these lethal genes are recessive. In Table XIV are listed some twenty conditions which have been reported as caused by lethal genes.

The Artificial Determination of Sex

For thousands of years human beings have desired the power of determining the sex of their children. This is not the place to consider the strange practices that have been resorted to and the fancy theories that even the most serious persons have entertained on this subject. Let it be sufficient to say of all of them that they were very wide of the mark.

Next to the power to be able to determine sex men have always wanted to be able to foretell the sex of their children after they have caused their wives to conceive. Since 1956 this has for the first time in the history of man become a scientific reality, and it is since 1957 that the determination of sex has become something more than a scientific possibility.

In 1956 Dr. Murray L. Barr of the University of Western Ontario discovered that from the earliest stages of development any cell of an organism destined to be a female could be distinguished from any cell of an organism destined to be a male. On the inside of the nuclear membrane of almost every female cell there is situated a structure called the *chromatin body* which takes a deep stain. In the cells of the male this body is not present. Hence, in order to be able to predict the sex of any human being immediately after

TABLE XIV
CONDITIONS REPORTED AS DUE TO LETHAL GENES

Recessive lethals

Acute idiopathic xanthomatosis (Niemann-Pick's disease). Great enlargement of spleen and liver with discoloration of skin	
Amaurotic idiocy	
(a) Infantile type	} Impairment of vision leading to total blindness, degeneration of nervous system, and idiocy
(b) Juvenile type	
Degeneration of the cerebral white matter	
(a) Acute infantile type	
(b) Subacute juvenile type	
(c) Convulsive type	
Epidermolysis bullosa	A skin disease in which blisters form on the slightest pressure
Gargoylism	Multiple growth derangement, gargoylelike face
Glioma retinae	Tumor of the retina
Ichthyosis fetalis	Scaling of the skin
Infantile muscular atrophy	Wasting of muscles with paralysis
Microphthalmia of the sex-linked type	Abnormally small eyes
Pseudohypertrophic muscular dystrophy	Muscular enlargement and paralysis

Semidominant lethals

Minor brachydactyly	Shortness of fingers
Pelger's anomaly	Unsegmented leucocytes
Sebaceous cysts	Cystic tumors of sebum-secreting glands
Spina bifida	Congenital cleft of vertebral column
Telangiectasia	Dilatation of capillaries, particularly serious nose bleeding

Adapted from Snyder, L. H., "The Mutant Gene in Man."

conception all one has to do is to obtain some of its cells, stain them, and examine them under a microscope. If they contain nuclear sex chromatin bodies then the developing embryo is going to be a female; if such bodies are absent the organism is almost certainly going to be a male. It is as simple as that. (See fig. 24.)

How does one obtain the cells of a living embryo or fetus *in utero*? At present this is done by a procedure called transabdominal amniocentesis, which means that the cells are obtained from the amniotic fluid, into which they have dropped from the organism surrounded by the fluid, by the passage of a needle through the mother's abdomen directly into the amniotic sac. This procedure has been practiced for other purposes, such as withdrawing excessive accumulations of amniotic fluid where its pressure has caused painful symptoms (polyhydramnios). This is not a procedure to be recommended, for it has sometimes resulted in ill consequences; we shall have to find some more satisfactory means of obtaining the cells of the developing uterine organism. We shall undoubtedly do so, but in any case we are now in a position to predict the sex of the human being long before he is born.

The discovery of the method of sexing by means of the chromatin body enables us to do for the first time, with virtually complete accuracy, many more things than the mere prediction of sex. For example, it now becomes possible for scientists to study the development from the fertilized egg of an organism whose sex is known even before there is the appearance in the embryo of the least external evidence of sex, which in man occurs at about the seventh week of development. Already great advances have been made in the study of developmental anatomy as a result. Furthermore, in many cases of true hermaphroditism, in which the individual exhibits the organs of both sexes, it is with a high degree of probability for the first time possible to determine the individual's genetic sex by taking some of the cells from his skin or the inside of his cheeks or any other part of his body and finding whether or not the nuclear chromatin body is present. This has already been done in many cases and constitutes a most important advance in what was hitherto a very difficult area. The technique has been used in medicolegal and forensic cases in the identification of fragmentary remains of bodies. Furthermore certain abnormal conditions which have hitherto been somewhat puzzling, such as Klinefelter's syndrome, are on the way to being considerably clarified. Klinefelter's syndrome is a condition in which "males," although they become masculinized, also develop small breasts as in the adolescent female (gynecomastia), and their testicles remain small and produce no spermatozoa, so that such "males" are sterile all their lives. It has been discovered that in all "males" characterized by Klinefelter's syndrome the sex

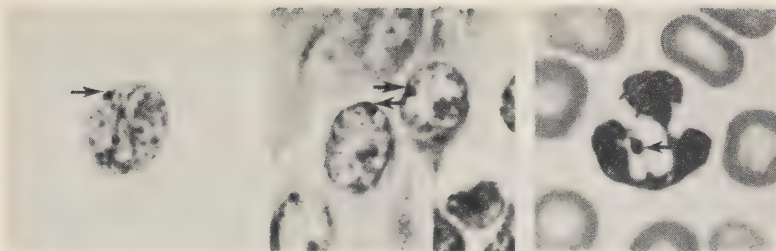


FIG. 24. The Chromatin Body in Female Somatic Cells. 1. Nucleus in an oral smear from a chromosomal female. 2. Cells of the spinous layer in a skin biopsy specimen from a chromosomal female. 3. Neutrophil in a blood film from a chromosomal female. (Courtesy of Dr. Murray L. Barr.)

chromosome number is abnormal. Yet these individuals, except for the adolescent feminine appearance of their breasts appear in every other way to be male.

It is a principal function of the sex chromosomes to direct the normal differentiation and development of the gonads (testes or ovaries). When for some reason this fails to occur this may result in a sequence of events that may lead to the phenotypical (the apparent) sex being the opposite of the chromosomal sex, as occurs in Klinefelter's syndrome and in the corresponding disorder in the "female" known as Turner's syndrome, in which there is a developmental failure of the ovaries with complete absence of ova—not to mention other associated bodily and psychological defects.

Studies published early in 1959 by four different teams of workers have conclusively shown that chromatin-positive Klinefelter's syndrome is associated with 47 chromosomes, the additional chromosome being an X chromosome; the sex chromosomal constitution of Klinefelter's syndrome is therefore XXY. Evidence is now also available that some cases of Klinefelter's syndrome are chromosomal mosaics, some cells bearing the normal 46 chromosomes, and others the abnormal number of 47.

Mongoloid idiots exhibiting Klinefelter's syndrome have been shown to possess 48 chromosomes, one additional autosome and one additional X chromosome.

In Turner's syndrome, on the other hand, C. E. Ford and his collaborators showed in 1959 that there are only 45 chromosomes present, and that such cases are of sex-chromosomal constitution XO.

The fourteen-year-old English girl exhibiting Turner's syn-

drome, described by Ford *et al.*, is anatomically and psychologically female, but nuclear-chromatin-negative, the latter being a condition usually associated with the male. Possessing only half the normal component of sex chromosomes, namely a single X chromosome, there is, however, no reason to assume that this girl is in any sense a male. Such an individual is not an example of sex reversal, but a female with an abnormal genotype, a female, however, who is an incomplete female—a female who would have developed as a completely normal female had she been endowed with another X chromosome, but who would have developed as a male had a Y chromosome been bestowed upon her. Thus, from cases such as this we learn that the hitherto enigmatic Y chromosome is the carrier of masculinizing factors.

There is considerable evidence that abnormal metabolism of maternal or placental hormones during pregnancy, when the gonads of the embryo are still undifferentiated, may produce sex reversal. Excessive amounts of progesterone or testosterone may produce masculinization of the female embryo so that it develops as a male or as a hermaphrodite or intersexual pseudohermaphrodite. In a recent study of 1,911 baby boys in England, Moore discovered that the sex chromatin was that of a genetic female in five cases. This is a frequency of 0.26 percent. It is probable that in some cases male to female sex reversal is produced in a similar manner. Intersexuality has been experimentally produced in the female progeny of pregnant rats who were injected with male sex hormones, androgens (A. R. Greene *et al.*). Similarly intersexuality appears to have been produced in some human genetic females by the administration to the mother during the first trimester of pregnancy of male sex hormones such as testosterone (Gold and Michael). Female to male sex reversal is often associated with mental defect. In a study of 663 prepubertal mentally handicapped English boys Ferguson-Smith found that eight of them, or 1.2 percent, were chromatin-positive females. Klinefelter's syndrome has recently been described in a set of seventeen-year-old identical twins. Holub, Grumbach, and Jailer consider that a genetic predisposition to develop this disorder probably exists.

In 1932 Dr. V. N. Schröder of the U.S.S.R. was able, by means of a simple electrical method, to separate X-bearing from Y-bearing spermatozoa. In this method sperm are placed in a solution,

the hydrogen-ion concentration of which can be controlled, contained in an electrophoretic apparatus. This apparatus has a positive electrode at one end and a negative electrode at the other. When an electric current is passed through the apparatus some sperm will migrate toward the positive pole (anode) and others toward the negative pole (cathode). It was found that the majority of sperm that migrate toward the positive pole are X-bearing while those that migrate toward the negative pole are Y-bearing. Dr. Schröder was able to predict in 80 percent of cases the sex of rabbits whose mothers had been artificially fertilized with such "positive" and "negative" sperm. In America, using similar methods, Gordon has been able to predict the sex of rabbits in 67.7 percent of cases. Sherry Lewin in England has reported similar behavior in human sperm. There can be little doubt that it will not be long before the artificial determination of the sex of human beings will become a practical possibility.

Dairy farmers may soon be able to produce all the heifer calves they desire and raise only enough bulls for the purposes of reproduction.

In the case of man what we need to learn is not so much how to determine sex as how to control the effects of sexual activity, more particularly both the quality and the quantity of human populations.

The Control of Population

In the year 1800 the total population of the world is estimated to have been about 906,000,000; in 1850 it was 1,171,000,000; in 1900 it was 1,608,000,000; in 1930 it was 1,987,000,000. Today it is about 2,750,000,000. By the year 2000 that figure will reach 6,250,000,000. Every second of the day three babies are born in the world, 180 every minute, almost 11,000 every hour, about 260,000 every day, well over 1,500,000 every week, and about 95,000,000 every year. If we go on at this rate, then by the year 2100 we may expect to have a world population of 10,000,000,000. This would give us a population of between 175 and 200 persons per square mile covering the whole face of the earth including the Antarctic Continent and the Sahara Desert. And after that, what then? Do we put on our space suits and migrate to other habitable planets? This deluge of popu-

lation has already seriously jeopardized the very existence of the human species, for it has brought in its train poverty, famine, economic convulsions leading to revolution and war, the desire of nations for more room—*Lebensraum*—and finally the hydrogen bomb as a means by which powerful nations can implement their expansionist intentions or protect themselves against such intentions by others, fancied or real.

Not only this, certain religions actually encourage their members to have many children regardless of the consequences to the families and the societies involved, for by this means of increasing their numbers such organized religions increase their political and social power. But it is not numbers that human beings should aim for but quality. Beyond a certain point, number itself greatly increases the difficulty of a problem. The size of cities and towns increases out of all proportion the difficulties of dealing in a humane and intelligent manner with human problems.

We now have not only the hydrogen bomb to contend with but also the population bomb.

This is not the place to consider whether or not the population will outrun the food supply—by technological means we may be able to meet the needs of even ten billion people, but that is not the real point. There are numerous other reasons than mere subsistence which should give us pause, for man does not live by bread alone. Human life is sacred and must be preserved. Every living human being should enjoy as his birthright the fullest opportunities for the development of his potentialities; instead, by the indiscriminate breeding which characterizes the human species almost everywhere on the face of the earth today we effectively deprive most human beings of their birthright, their sacred right to development, and we jeopardize the chances of all but a fortunate few to achieve that development.

We should not be so much concerned about the increase in the number of mentally inadequate persons, the otherwise deficient, and the hereditarily disordered as we should be about the total increase in the number of human beings. We have nothing to fear from the inadequate—it is the increase, beyond what is good for the survival of the species as a whole, of the humanely undeveloped that we have most to fear. Certainly we should always be concerned with the quality of human beings, and the best way to do so is to see to it that the sum of human beings is kept within controllable dimensions. First things should come first.

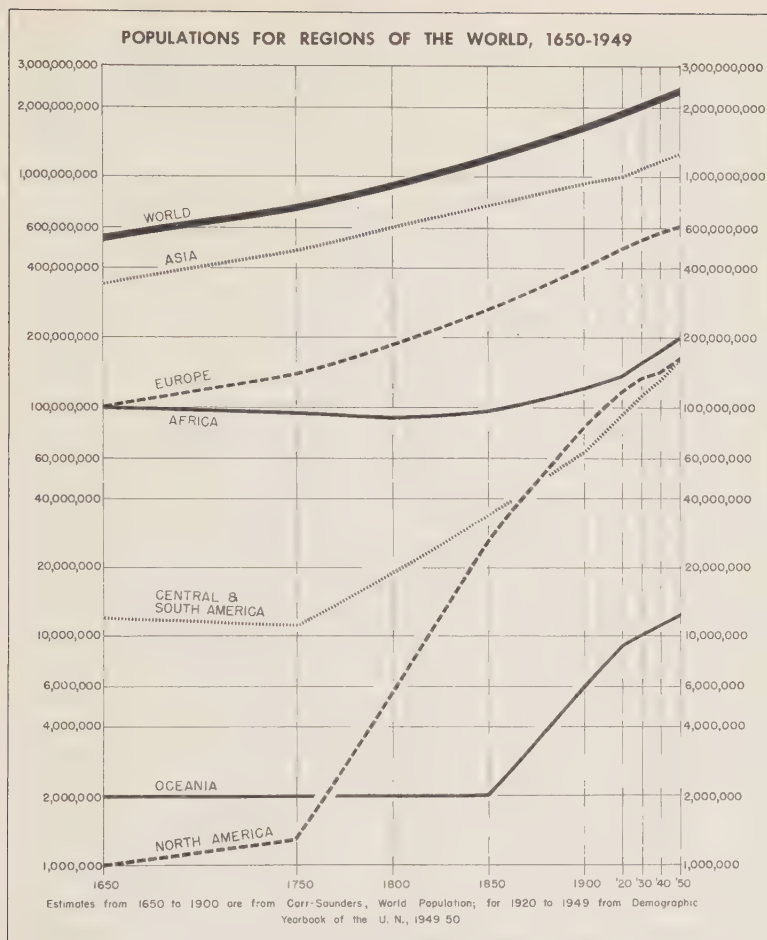


FIG. 25. World Population. (Courtesy of U.S. Public Health Service.)

The birth rate has not appreciably risen in the last fifty years. What has served to produce the tremendous increase in population is the lowering of the death rate. In India the excess of births over deaths has trebled within the last thirty years. Medical and sanitary advances in the control of disease have been chiefly responsible. But as William Vogt has asked, "Is there any kindness in keeping people from dying of malaria so that they can die more slowly of starvation?" Professor Conway Zirkle has asked, "Have

we who believe in birth control a moral right to send food to peoples who don't?" These are very real questions, but however they are answered, it is certain that the conditions which caused these questions to be asked at all should never have been allowed to arise. But they *have* arisen, and they are very much with us. What are we going to do about them? In other words, What are we going to do about the population problem? "The whole human race is rumbling on to destruction," declares Lord Boyd-Orr.

The present methods of limiting fertility, the Malthusian quartet—war, famine, disease, and voluntary control—are not good enough. In improved methods of voluntary control, however, lies the only hope of our being able to do something about limiting the explosive rate of uncontrolled human multiplication. If we had only a small fraction of the scientific personnel working on this problem who are now devoting their energies to the development of more destructive armaments, we should make great progress in a very short amount of time. What are needed are oral contraceptive pills which will work directly on the ovary, or the uterus, or the fallopian tubes, that is to say, which will either temporarily stop ovulation or make implantation of the fertilized ovum impossible in the uterus, or close off the fallopian tubes from the uterus. Research along these lines is proceeding, but there are only a comparative handful of scientists at work on these problems. Already pills have been tried that work on the pituitary gland which have proven quite effective in the control of conception. Such pills cost from thirty to fifty cents each, but it is too early yet to say whether pills that work directly on the pituitary are really desirable as contraceptives. Should they be shown to have no side effects, these pills will perhaps constitute the first genuinely significant contribution to the control of conception throughout the world, and will thus be the first major step to be realized in the control of population. The first step toward the control of the quality of population is to control its quantity.

Telegenesis or Long-Term Artificial Insemination

Men have often thought how wonderful it would be if we could preserve the sperm of great men and then artificially inseminate with them selected women of choice heredity and in this way increase the chances of producing more great men and women like their progenitors. Thus far, this dream has been realized in the

pages of science fiction, among the most notable nightmarish realizations of which is contained in Aldous Huxley's *Brave New World* with its Central Hatcheries and Conditioning Centers and babies who are decanted from glass bottles. Huxley's book was published in 1932. We still have a little way to go before we can decant human babies from bottles, but it can be done with some lower animals, and there can be little doubt that it will be achieved with human beings. Meanwhile, however, the dream of being able to inseminate women with the spermatozoa of men long dead is in process of realization. Experiments show that the spermatozoa of bulls may now be preserved for several years at the temperature of dry ice, -79°C. , in a special glycerol-containing medium and thawed out for use in inseminating cows. At the present time there are thousands of calves who have already been born from such artificial inseminations, and from fathers, in many cases, who have been dead for some time before they became fathers!

Interestingly enough, human spermatozoa are much better able to withstand low temperatures than are the spermatozoa of other mammals. It has been possible to preserve human spermatozoa by slow freezing to -79°C. in semen and glycerol, and by subsequent thawing for most of the spermatozoa to recover their motility without the loss of more than one third. Drs. Bunge and Sherman of Iowa City have carried this work to its logical conclusion and by means of spermatozoa preserved in this manner succeeded in inseminating three women who became pregnant as a result and produced perfectly normal children. From the experience with domestic animals there is not the least reason to think that the spermatozoa exposed to these low temperatures for long periods of time have in any way suffered from the experience.

We see then that the long-term preservation of human spermatozoa in a thoroughly efficient condition is now a practical possibility, and that long-range posthumous paternity is now probably simply a matter of improving the means of preserving spermatozoa. Nor can there be much doubt that when our scientists have adequately addressed themselves to the task of preserving whole ovaries with their thousands of ova, they will succeed in doing so, and that we can therefore look forward to long-term posthumous maternity as well! The control of human heredity is increasingly being placed within man's own power. This is a subject we shall deal with in Chapter 17. Meanwhile, let us proceed to the discussion of heredity and "race."

Hereditv and "Race"

"**R**ACE" HAS BEEN put between quotes at the head of this chapter because it is a questionable term. Most persons who use the term think they know what it means, and what most persons mean by the term is not in agreement with the facts. The idea that "race" means that certain peoples, the so-called "races," are characterized by definite inborn physical and mental traits is widespread. It is an understandable enough error. One sees a whole people who look physically unlike any other group of people; they often speak a unique language, or if they don't they may speak a language in a peculiarly distinctive manner; their customs are different from any other people, and they behave in ways that differ from those of other people. Taken together, all these things distinguish this group of people from all other peoples in the world. And this is what is meant by "race." If that is, indeed, what most people mean when they use the word "race," then the word is beyond rescue and it had better be dropped altogether, for its use in the above sense only serves to perpetuate an amalgam of errors which obscures the truth and serves to erect barriers between peoples where such barriers do not, in fact, exist.

The principal error committed is the assumption that the observed physical differences are innately linked with the observed behavioral differences, and hence the behavior as well as the physical differences are said to be due to "race." The Germans, the French, the English, the Jews, the Moslems, the Negroes, the Chinese, the Japanese, the Italians, and the rest all behave the

way they do, it is commonly believed and maintained, because they belong to different "races." Altogether apart from the fact that none of the groups mentioned, even in the strictly biological sense in which the term might be used, constitute "races," it is a common habit to attribute to "race" any form of behavior that is different, and the physical or other characters of the other group provide a convenient peg upon which to hang the differences. Instead of making the effort to understand the meaning of the differences, we too often tend to dismiss them as "inferior," "undesirable," "crude," "repugnant," or at best as "quaint" or "queer." And often enough we settle for what seems to us the inescapable conclusion that we are members of the "superior" group and the others are members, of course, of the "inferior" group. This has always been a comforting decision at which to arrive, for it at once secures one in the belief that one is better than other people and absolves one from the necessity of further inquiry, for are not the facts self-evident? Unfortunately, too often what is evident in facts that are "self-evident" is not the facts but one's self, the self that gets in the way of the facts, distorts them, and prevents us from seeing them as clearly as we otherwise might. Let us then consider the facts. A simple and effective technique in dealing with words that contribute to muddled thinking is to drop them altogether. Because the term "race" closes the door on what we are trying to understand, let us use instead the noncommittal term "ethnic group." By the use of such a term we commit ourselves to no particular theory, but in effect say that what the differences between ethnic groups may represent is a matter for inquiry rather than for closed judgment, that if we are to obtain a sound view of the facts we must keep our wits sharpened and our minds open. When we have satisfied ourselves about the facts, we can then decide whether to use one term or the other. The word "ethnic" is derived from the Greek *ethnos*, meaning a nation, people, tribe, or group.

Heredity, Ethnic Group Characters, and the Evolution of Man

We see clearly that some people are black-skinned, that they have broadish noses, smallish ears, black kinky hair, thickish lips, and relatively little body hair. We call such people Negroids. Others have straight black hair, a tendency to projecting upper

front teeth, a fold of skin over the inner angle of the eye, and the barest suggestion of a yellowish tinge to the skin. We call such people Mongoloids. Still others have a white skin, a long narrowish nose, all sorts of varieties of hair color and hair form, and a tendency to develop much body hair. Such people we call whites or Caucasoids. And there are a large variety of other groups—no one knows how many—each with their special assemblage of physical traits.

What is the meaning of all these physical differences among human groups? Are they possibly caused by the fact that all the ethnic groups of man are descended from different kinds of monkeys and apes? This question has been seriously considered in the light of the relevant evidence, and it is generally agreed that all the ethnic groups of man must have originated from a single ancestral stock closely resembling, but considerably more advanced than, the extinct manlike apes from South Africa known as the Australopithecinae. These were a group of erect-walking apes with the most manlike teeth of any known manlike ape. The australopithecines were probably near relatives of the stock from which man originated. The members of all ethnic groups are far too much alike in their structural and functional characteristics for them to have originated from different apelike forms. And that is precisely the point, the more we study the different ethnic groups of man the more alike they turn out to be. The likenesses by far outnumber the differences.

If, then, all the ethnic groups of man originated from a single ancestral stock, how did the differences we now observe among them come about? The answer to this question is, Probably in much the same way as the differences we observe among different varieties of the same species of any wild animal.

The human species, *Homo sapiens*, is about a million years old. This gives us plenty of time for the development of ethnic group differences. Human populations were very small in the prehistoric period and can rarely have consisted of more than several hundred individuals. Let us commence with a group of a few hundred individuals. After some time the food supply begins to diminish and a number of families decide to try their lot and *migrate* over the range of mountains. They do so and settle in a region with a good food supply. From this population another group of families decides to splinter off and go seek their fortune beyond the forest.

Another group of families decides to cross the water and settle on the island which they can see in good weather. And so it happens that in the course of time migrating groups of human beings would form settlements, in which they would often remain *isolated* for thousands of years from all contact with other people. Since *mutations* would occur in individuals within each group, and since mutations are always random, some of these fortuitous mutations would gradually establish themselves in the small breeding isolate, and in this way every separated group would in the course of time come to differ physically from every other—that is to say, genotypically in the frequencies of certain genes and phenotypically in the distribution of certain physical traits. Some of the mutations that would establish themselves would undoubtedly be of adaptive value to the members of the group. For example, those possessing a large amount of dark pigment in their skin in a climate in which the sunlight intensity was high would be likely to enjoy a selective advantage over those with lighter skins. The dark-pigmented individuals would tend to leave a larger progeny behind them. In other regions where the sunlight intensity was low and the skies often cloudy, more lightly pigmented skin would be an advantage in more freely allowing the rays of the sun to penetrate the skin; also fewer sweat glands would be necessary. This sort of development would be the factor of *natural selection* or adaptive fitness at work. Since it is highly improbable that the same kinds of mutations would have occurred in different isolated groups, it will readily be understood how on this basis alone different human groups would come to differ from one another in the course of time. Some of the mutations would be useful, and others would simply become randomly fixed within the group even though they might have no particular adaptive value; such a process is known as *genetic drift*. In the course of time some of the members of these groups might meet, form permanent unions, *hybridize*, and create new settlements. By producing new gene combinations, and allowing for the action of selection, isolation, genetic drift, etc., an altogether new physical type would be formed.

The operation of the six factors mentioned, *migration*, *isolation*, *mutation*, *genetic drift*, *natural selection*, and *hybridization*, is sufficient to account for the evolution of the genotypic and phenotypic differences which distinguish the ethnic groups of man.

Genetically speaking, an ethnic group is a population differing in the frequency of some gene or genes which is actually exchanging or is capable of exchanging genes across whatever boundaries separate it from other populations of the species. All living mankind is included in the single species *Homo sapiens*, a species which consists of a number of populations which individually maintain genetic and phenotypic differences from one another by means of isolating mechanisms such as geographical and social barriers. In addition to the effects of other influences, these differences will vary as the power of the isolating mechanisms vary. Where these barriers are of low power, neighboring isolates will integrate or hybridize with one another. Where these barriers are of high power, such isolates will tend to remain distinct or replace each other geographically.

Such isolates constitute the *ethnic groups*, which may be defined as arbitrarily recognized groups that in virtue of the possession of a more or less distinctive assemblage of physical traits, derived through a common heredity, are statistically distinguishable from other groups within the species.

It is observed that most of these ethnic groups tend to form certain clusters according to their resemblances in certain characters. For example, black skin yields a whole cluster of Negroid groups; white skin yields another cluster of white or Caucasoid ethnic groups, and yellowish skin the cluster of Mongoloids; while chocolate-brown to brownish-white skin associated with abundant, wavy head hair yields the Australoid or "archaic Caucasoid" cluster. These clusters of characters suggest that some of the ethnic groups exhibiting them may be, in respect of these characters, more closely related than they are to the members of other clusters. Such clusters are termed "major groups."

A *major group* comprises a number of ethnic groups classified together on the basis of their possession of certain common characters which serve to distinguish that major group from others. Four such major groups as stated above are recognized. The usual classification is set out in the diagram in figure 26.

Ethnic and major group differences simply represent more or less temporary expressions of variations in the relative frequencies of genes in different parts of the species range. Such a conception rejects altogether the all-or-none conception of "race" as a static condition of fixed differences. It denies the unwarranted assumption that there exist any hard and fast genetic boundaries



FIG. 26. The Major Ethnic Groups of Man, Showing the General Distribution of Various Traits. (From Ashley Montagu, *An Introduction to Physical Anthropology*.)

between any groups of mankind and asserts mankind's common genetic unity in diversity. Such a conception of the variety of man cuts across national, linguistic, and cultural boundaries, and thus asserts the essential independence of genetic factors.

The Fundamental Genetic Likeness of Mankind

The differences that distinguish the ethnic groups are real and important, important in the sense that they are adaptively valuable. But even between ethnic groups with extreme differences in appearance, it is unlikely that the number of gene differences exceed more than a fraction of 1 percent.

Were an anthropologist to be asked to name the two ethnic groups who physically appear to be most unlike each other, he might mention a Congo Pygmy and a Scandinavian white, or he might mention a white European and a South African Bushman. In fact, these appear to be such extreme types that a learned German professor, Fritz Lenz, not so long ago declared, "As far as I am aware, neither African Pygmies nor Bushmen interbreed with Negroes or with Europeans; thus, owing to their natural instincts and their habits, they are physiologically isolated." How wrong can a learned German professor be! Neither the "natural instincts"—whatever they may be—nor "their habits" have prevented such interbreeding from occurring. We do not, however, have adequate reports on Pygmy-Negro offspring, but we do have an adequate report on Bushman-European interbreeding. Such a report was published in 1954 by Dr. Phillip Tobias of the University of Witwatersrand, South Africa. Even though only three generations of a single family are involved, this report throws much light upon the genetic likeness of the ethnic groups of man.

In the first generation of this family, a Bushman woman, shown in figures 27(1) and 27(2) mated with a Dutch white, who was not available for study. The offspring of this union was a daughter, shown in figures 27(3) and 27(4) who presented an appearance intermediate between her Bushman mother and her white father, but with most of her physical characters, which were determined by measurement and inspection, much more closely resembling her white father than her Bushman mother (see photographs). The hybrid daughter was $4\frac{3}{8}$ inches taller than her mother, her face was wider and longer, her eyes more closely set, her nose narrower and higher. The hair was long, brown, wavy, and of silken texture, whereas her mother's head hair was of the tightly coiled and tufted variety known as "peppercorn" typical of the Bushman. Eyebrows and eyelashes were well supplied with hair, and



1



2



3



4



5



6



7



8

FIG. 27. 1. Bushman mother, from in front, and (2) in profile. 3. Hybrid daughter of white father and Bushman mother, from in front, and (4) in profile. 5. Daughter of white father and hybrid mother shown in 3, granddaughter of woman shown in 1, from in front, and (6) in profile. 7. Husband of hybrid shown in 3 and father of their daughter shown in 5, here with their daughter. 8. Bushman mother, hybrid daughter, and hybrid granddaughter. (Courtesy of Dr. P. V. Tobias.)

the body hair was moderately profuse in contrast with the mother's relative hairlessness. Her eyes were brown and her skin light. The ears were without lobes as in the mother. Eye folds (similar to those seen in many Chinese and Japanese) are present in the mother and daughter, which suggests that both the eye fold and lack of ear lobes behave as dominants. The daughter is in general of much larger and broader build than her mother.

The daughter formed a union with a white Englishman shown in figure 27(7), and by him had two sons and two daughters. The youngest son died at an early age from the bite of a button spider. According to reliable witnesses, the remaining son looks European, one of the girls looks more like her mother than her father, and the only child available for study, the three-year-old girl shown in figure 27(5) looks in every way like a European child. This little girl has light brown eyes, very fair skin, large ears possessing a lobe, and she has a definite eye fold; the nose is narrow and high, but it has a slightly bulbous tip as does that of her mother and grandmother. Thus, the only features this little girl has in common with her Bushman grandmother are the eye fold and bulbous nose tip. This girl's sister resembled her in being of distinctively European appearance, though her surviving brother, who was not seen and who was European-looking, was said to show rather more evidence of his Bushman grandmother's genes. This is a curious fact, namely, that more European features should appear in the females than in the male. This observation has been recorded for other crossings and is apparently the result of sex influence acting differentially upon the genes involved. But our main point here is the demonstration of the fewness of the gene differences existing between human types that appear extremely different from one another. Were the differences more numerous than they are, we should expect to see more marked differences in the hybrid offspring of such unions, and we should certainly not expect to see virtually all the traits of a white grandfather and father segregating out as they have done in this little girl whose grandmother was a pure Bushman and whose mother was a hybrid of Bushman-white origin. Nor would we expect to see a first-generation hybrid such as the mother so remarkably white and with so few Bushman characteristics if there were many significant gene differences either in number or kind between Bushman and white. The evidence strongly indicates that man-

kind has obtained the vast majority of its genes from the same common gene pool, and that such mutational differences as have occurred in the course of the evolution of the different ethnic groups have, in comparison with the number of genes that they hold in common, been relatively few.

Is "Race"-Crossing Harmful?

It has often been asserted that "race"-crossing is harmful: that it succeeds in perpetuating the worst traits of both "races" in the hybrid, that it produces physical disharmonies, that "race" mixture causes contamination of good stock, that it produces an inferior generation, and so on. All these statements are quite unsound even though they are widely believed. Since half-castes have been more often than not treated as outcasts by society and at best have occupied an anomalous and ambiguous position in society, it is not to be wondered at that they have frequently been regarded by those who forced such a degrading position upon them as "inferiors" or "degenerates." But their "inferiority" has seldom if ever been biological, but rather a social inferiority that has been thrust upon them and justified by the myth of their alleged biological inferiority. The disparities in the social attainments of half-castes or hybrids and whites can be explained by the difference in opportunities as well as abilities. The evidence is full to overflowing on this point in the United States where Americans of white and Negro origin have earned positions of respect and honor in their communities.

Unfortunately a considerable amount of prejudice has been the rule in this area of conversation, and even scientists have not been guiltless of it. An outstanding example of this is the case of the late Dr. C. B. Davenport, one of the fathers of the science of genetics in America and a distinguished scientist. In a study entitled *Race Crossing in Jamaica*, published by the Carnegie Institution of Washington, D.C., in 1929, in which Davenport interpreted the findings of the field investigator, Dr. Morris Steggerda, on "Black," "Brown," and "White" Jamaicans, the "Browns" being the crosses of the "Blacks" and "Whites," one can perceive Davenport's prejudices at work quite amazingly. Here, for example, is one of the most extraordinary illustrations of such prejudices at work: "The Blacks," writes Davenport on

page 469, "seem to do better in simple mental arithmetic and with numerical series than the Whites. They also follow better complicated directions for doing things. It seems a plausible hypothesis, for which there is considerable support, that the more complicated a brain, the more numerous its 'association fibers,' the less satisfactorily it performs the simple numerical problems which a calculating machine does so quickly and accurately." In other words, when the "Blacks" do better than the "Whites" their superiority must be explained away! And not only explained away but cited as a further evidence of their inferiority!

Some of the hybrids measured by Steggerda exhibited a combination of "long arms and short legs." Upon this, Davenport commented as follows: "We do not know whether the disharmony of long arms and short legs is a disadvantageous one for the individuals under consideration. A long-legged short-armed person has, indeed, to stoop more to pick up a thing on the ground than one with the opposite combination of disharmony in the appendages."

Three out of four brown (hybrid) Jamaicans are cited in support of this generalization. Here are the figures upon which this generalization is based:

Limb Proportions and Stature in Jamaicans

	<i>Black</i>	<i>Brown</i>	<i>White</i>
Arm length in cm.	57.3	57.9	56.8
Leg length in cm.	92.5	92.3	92.0
Total stature in cm.	170.6	170.2	172.7

From these figures it will be observed that the arm length of the browns, that is the hybrids of crosses between the whites and blacks, is, on the average, 0.6 centimeter greater than in the blacks and 1.1 centimeter greater than in whites, while the leg length of the browns is 0.2 centimeter less than in the blacks. It is here that the "disharmony" is perceived by Davenport. It has, however, to be pointed out that the order of the differences in total stature is so small—at most not more than 2.5 centimeters (1 inch) between brown and white—while the average difference between length of arms and legs among the three groups never exceeded more than half an inch, that it could not make the slightest practical difference in the efficiency of stooping.

As the distinguished doyen of genetics in America, William E.

Castle, has said, "We like to think of the Negro as an inferior. We like to think of Negro-white crosses as a degradation of the white race. We look for evidence in support of the idea and try to persuade ourselves that we have found it even when the resemblance is very slight. The honestly made records of Davenport and Steggerda tell a very different story about hybrid Jamaicans from that which Davenport and Jennings* tell about them in broad sweeping statements. The former will never reach the ears of eugenics propagandists and congressional committees; the latter will be with us as the bogey men of pure-race enthusiasts for the next hundred years."

The type of evidence that is sometimes wrongly interpreted by investigators as evidence of the ill effects of "race"-crossing is illustrated by a study made by Miss R. M. Fleming in 1939. Studying the offspring of Negro-white unions in the seaports of England and Wales, Miss Fleming found that 10 percent of the hybrids showed a disharmony between the teeth and jaws. The palate was generally well arched, while the lower jaw was V-shaped and the lower teeth slipped up outside the upper lip, seriously interfering with speech, this disharmony "resulting where a well-arched jaw was inherited from the Negro side and a badly arched one from the white side." No other disharmonies were observed.

Miss Fleming states that a "badly arched" jaw was inherited from the white side. In other words, the disharmony was not due to the effects of crossing but to the fact that the condition was transmitted from the white parent to the child. The disharmony was limited to only 10 percent of the cases. It is also possible that while some of these cases merely represent the expression of inherited defects, not necessarily exhibited in the jaws of the parents themselves, still others were due to malnutrition and that the defect actually bore no relation whatever to the fact that one parent was a Negro and the other a white. Otherwise, it would be expected that more than 10 percent of the hybrids would exhibit disharmonies of the relations of the jaws to one another.

As far as the evidence goes there is not the slightest reason to suppose that "race"-crossing ever produces disharmonies of any

*Professor Herbert S. Jennings in his book *The Biological Basis of Human Nature* adopted Davenport's interpretations of the ill effects of hybridization, and it is to this that Professor Castle refers.

kind. The fact seems to be that the differences between human groups are not large enough to be capable of producing even the slightest disharmonies.

For the same reason there appears to be very little evidence of *hybrid vigor* as a consequence of crossing between ethnic groups—the gene differences do not seem to be large enough. Hybrid vigor is the phenomenon observed in plants and animals where, as a result of the crossing of different varieties, offspring tend to be more fertile, numerous, stronger, and larger than the members of the parental stock.

The principal determining factor in the physical organization of the new human being, the offspring of any union, is the genetic constitution of the parents, and nothing else. Since the evidence leads us to believe that no human ethnic group is biologically either better or worse in any trait, group of traits, or as a whole, than any other, it should be clear that hybridization between human beings cannot lead to anything but a harmonious biological development.

In emphasizing the basic essential likeness of all the ethnic groups of man, let us not forget that fundamental and important as these are the really valuable qualities that human beings exhibit are not their likenesses but their differences. It is these very differences—the physical differences between the ethnic groups of man—evolved over the long course of human history, that constitute the best testimony to their own value, for it is these physical differences that have enabled the ethnic groups to survive in the environments in which they originated. Hence, there can be no question of “superiority” or “inferiority” in the comparison of these physical ethnic traits; all of them constitute evidence of adaptive fitness, and the genotype of man is such that it is capable of making very rapid phenotypic adjustments to every conceivable environment to which it is called upon to respond. The members of any ethnic group can live in any environment in the world, and by resorting to intelligence and imagination they can adjust to the widest extremes of climate, or any other conditions they are called upon to meet.

“Race” and Mental Capacities

The notion that “race” represents “something” which is an amalgam of physical and mental traits, so that certain physical

traits go together with certain mental traits, is widely held. Indeed, this is the core of what most people understand by "race." In spite of innumerable attempts to prove that such a linkage exists between physical and mental traits no one has succeeded in demonstrating it or even reasonably indicating that such a linkage exists. This is not to deny that some slight differences may exist between some ethnic groups in the frequencies of certain genes underlying mental capacity. It is possible that such slight differences exist, but in spite of all attempts no one has, in fact, ever demonstrated that they do. But one thing seems to be highly probable, and that is if such slight differences exist, then they in no way depend upon physical traits. The evolution of the physical traits of ethnic groups has been in adaptation to totally different kinds of environmental challenges than those that have been involved in the evolution of man's mental capacities.

While the challenges of the physical environments have been very different in different geographic areas of the world with consequent differences being established in the patterns of genotypic adaptations observable in the physical differences between ethnic groups, the challenges of the many social environments have been fundamentally similar. If we ask ourselves on what qualities in every society, at any time level, the highest premium was put, and we check this by the facts, we find: the ability to get along with people, the ability to know when to say yes and when to say no, when to keep one's mouth shut whatever one may want to say, the ability to make rapid adjustments to rapidly changing conditions, to know when to stay and fight or when to run away and live to fight another day—in other words, the qualities of maturity of judgment, wisdom, and adaptability. It is this combination of traits, adding up to what we can call the trait of plasticity, which appears to have been at a high premium in all societies—as it still is. This plasticity is not a particular trait but a general one. Instead of leading to fixed responses to the environment, man's evolution has been such as to make him the least behaviorally fixed and the most generally educable or plastic of all living creatures. It is this very plasticity of his mental traits that confers upon man the unique position that he occupies. The acquisition of this capacity freed man from the constraint of the limited range of biologically predetermined responses that characterizes all other animals. In the history of life on this earth man is the only creature who became capable of substantively

controlling his physical environment instead of being controlled by it. He began to live his own life instead of being lived principally by his organic limitations. In all times and in all climes the process of natural selection seems to have favored genotypes which permit greater and greater educability and plasticity of mental traits under the influence of the uniquely social environments to which man has been continuously exposed.

The effect of natural selection in man has probably been to render genotypic differences in personality traits, in mental traits, in genetic potentialities, between individuals and particularly between ethnic groups, relatively unimportant compared to man's phenotypic plasticity. Man's genotype is such that it makes it possible for him to develop the widest range of behavioral adjustments and adaptations. Instead of having his responses genetically fixed as in other animal species, man is the species that invents its own responses, and it is out of this unique ability to invent, to improvise his responses that his cultures are born.

Examining the end effects of the long story of human evolution as they present themselves today—namely, human beings of every ethnic group—the conclusion seems inescapable that natural selection has been operative upon the traits of educability and plasticity in much the same way from the beginnings of man's history, in all human groups, no matter how long isolated they may have been from one another. What, in short, has been at the highest premium in all societies is not some special ability but rather the general ability of behavioral plasticity. Natural selection has favored and continues to favor plasticity. Genetic differences between individuals are by no means washed out, but those differences are retained only if they are of the kind that permit themselves to be eclipsed by phenotypic plasticity. Hence, it is not to be expected that there exist any very significant differences in the distribution of special abilities among the ethnic groups of man.

"Race" and Intelligence

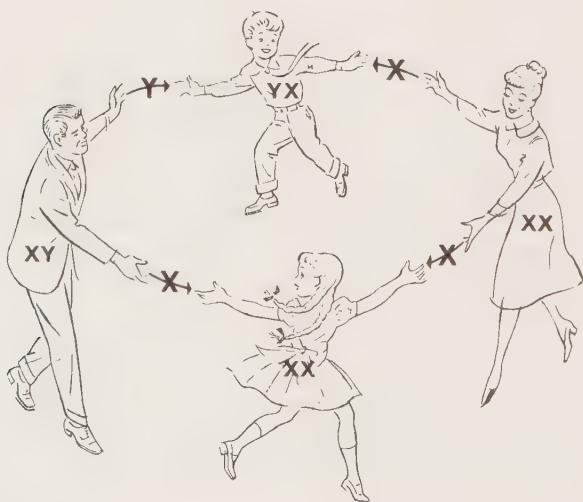
If the points we have made in the above section are sound, then there should exist few if any really significant differences in the genetic capacities for intelligence of the different ethnic groups. In spite of many attempts to demonstrate that such differences

in intelligence exist, there has been complete failure to do so. Certainly differences in intelligence as measured by performance have been found to exist between the ethnic groups of man, but it is one thing to find such differences and quite another to demonstrate that they are due to genetic factors. On the other hand, when it has been possible to demonstrate anything whatever in relation to the intelligence tests on different ethnic groups, it has consisted in the finding that when the environment is equalized the performance on the intelligence tests becomes equalized.

Intelligence is a function not alone of genes or of environment alone but of the interaction between both. Intelligence implies not only innate potentialities but opportunities for the realization of those potentialities. What applies to different cultures applies to the members of them. Why is it that the Australian aborigines differ in cultural attainments as much as they do from Europeans? Is it because of the differences in the qualities of the intelligence of aborigines and of Europeans? The answer is almost certainly no. The answer is that the differences in the cultural achievements of Australian aborigines and, say, Englishmen is entirely due to the difference in the history of the cultural experiences to which each has been exposed. It should be recalled that until the eighteenth century the Australian aborigines were an undiscovered people who had been isolated for thousands of years on one of the southernmost islands of the world, and that they had had practically no contacts whatsoever with the outside world. The greater part of the territory occupied by them was (and is) one of the most inhospitable desert regions of the world, and to it they have made a perfect adjustment. What, in comparison, has been the history of the English, or for that matter, any other European nation we wish to consider? It has been a history of continuous cultural contacts with numerous other peoples over several thousand years in an area of the world where the cross-fertilization of ideas, customs, and ways of life have been of the most stimulating kind. Even today, the Australian aborigines have been largely prevented from realizing their magnificent potentialities by discriminatory practices which are not unknown elsewhere in the world. But when Australian aborigines are given the opportunity they show themselves capable of achievement at least as great as that of the average man anywhere else. Some fifty years ago there was a school in the southwestern part of Aus-

tralia, in the state of Victoria, which was attended entirely by aboriginal children. I quote the Reverend John Mathew: "In schools it has often been observed that aboriginal children learn quite as easily and rapidly as children of European parents. In fact, the aboriginal school at Ramahyuck, in Victoria, stood for three consecutive years the highest of all the state schools of the colony in examination results, obtaining *one hundred per cent. of marks*." (The italics are the Reverend Mathew's.) The extraordinary artistic ability of the Australian aborigines is only a very recent discovery, for as soon as they were taught to see as Europeans do, both children and many adults soon greatly surpassed in their artistic performances anything that the average European child or adult was able to do.

The story is everywhere the same, as we have already seen on an earlier page; wherever and whenever individuals are given the opportunities to realize their potentialities, we find that human beings everywhere can do what other human beings anywhere have done. Of course there are great variations in the distribution of abilities in every population, but the range of these differences is far greater *within* each ethnic group than *between* different ethnic groups.



PART II

The Family Album

*The face in the family album
Is the face you have always seen,
But it isn't the face of the thousands
Of faces you might have been.*

A.M.

In the second part of this book we shall consider the heredity of common physical and functional traits. In some cases the hereditary mechanism is known, in others not.

The Pigmentary System of the Skin, Eyes, and Hair

THE SKIN is the largest organ in the human body and one of the most complex. In addition to its functions of protection, respiration, thermal regulation, sensory and motor response to stimulation, the skin manufactures ergosterol, which is the provitamin of vitamin D, so important in the growth of bones and the development of teeth. To assist it in the execution of its functions the skin is more or less deeply pigmented. The principal pigment, called melanin, originates in a deep layer of the skin known as the germinative layer and then passes into the adjacent upper layer known as the granular layer of the skin or epidermis. The amount of pigment in the skin is inherited, but it is not known precisely how many genes are involved, nor is the hereditary mechanism understood. Anything from two to twelve genes have been suggested as involved in skin color, but the truth is that we do not know how many are in fact involved. There must be many. We shall return to this matter again. What we do know with certainty is how, for example, the condition known as *albinism* (Latin *albus*, white), in which there is a complete lack of pigment in the skin, hair, eyes, and nails, is inherited.

Albinism

Albinism is believed to be due to a biochemical block resulting from the lack of a particular enzyme acting on the tyrosine which through a series of biochemical steps leads to the formation of

melanin, the pigment of our skin, eyes, and hair. The skin is white, the hair is straw-colored in appearance, and the eyes look pink owing to the reflection of the blood vessels in them. Albinism is known to occur in whites, Negroes, American Indians, and probably occurs in other ethnic groups. It is very common in mice, rats, and rabbits, and has been reported in many different kinds of animals.

In man about 1 out of every 20,000 children born is an albino, and in a large proportion of cases the birth of such a child occurs in family lines in which there has never been a previous birth of an albino. In some cases the albino birth may be due to a mutation, but more often than not it is demonstrably due to the coupling of recessive genes for albinism, one derived from the mother, the other from the father, both of whom are carrying them in their heterozygous recessive state. Such a couple discovering that they are heterozygous for the albino gene stand a 1-in-4 chance of producing another albino child. The chances of any couple picked at random bearing an albino child are 1 in 20,000. This is a rather surprising figure in view of the fact that 1 out of every 70 persons carries the gene for albinism. Actually the heterozygous carrier is 286 times more frequent than the homozygous albino.

Now, if 1 out of every 70 persons is heterozygous for albinism why are there so few albinos, only 1 out of every 20,000? The answer is a matter of simple arithmetic. The chance of any man carrying the albino gene is 1 in 70, the chance that the woman he marries carries the gene is also 1 in 70. The chance that both carry the gene is 70 times 70, that is, 1 in 4,900. Finally, the chance that the child of such heterozygous parents will be an albino is 1 in 4, and 4 times 4,900 is 19,600, which give us the approximate estimate of 1 in 20,000 being born an albino.

The frequency of 1 out of every 4 children of a heterozygous couple standing a chance of being an albino derives from the following considerations. Let *C* stand for the dominant gene for normal pigmentation, and *c* for the recessive gene for albinism, the heterozygous male carrier then has the genotype *Cc* in his body cells while half his sperm carry *C* and the other half *c*. The same will be true of the heterozygous female and her eggs, half will carry *C* and half *c*. If you will take a pencil and paper and try to figure out the possible combinations into which each of these genes from sperm and egg can enter, you will find that there will be one *CC* child, two *Cc* children, and one *cc* child who will be al-

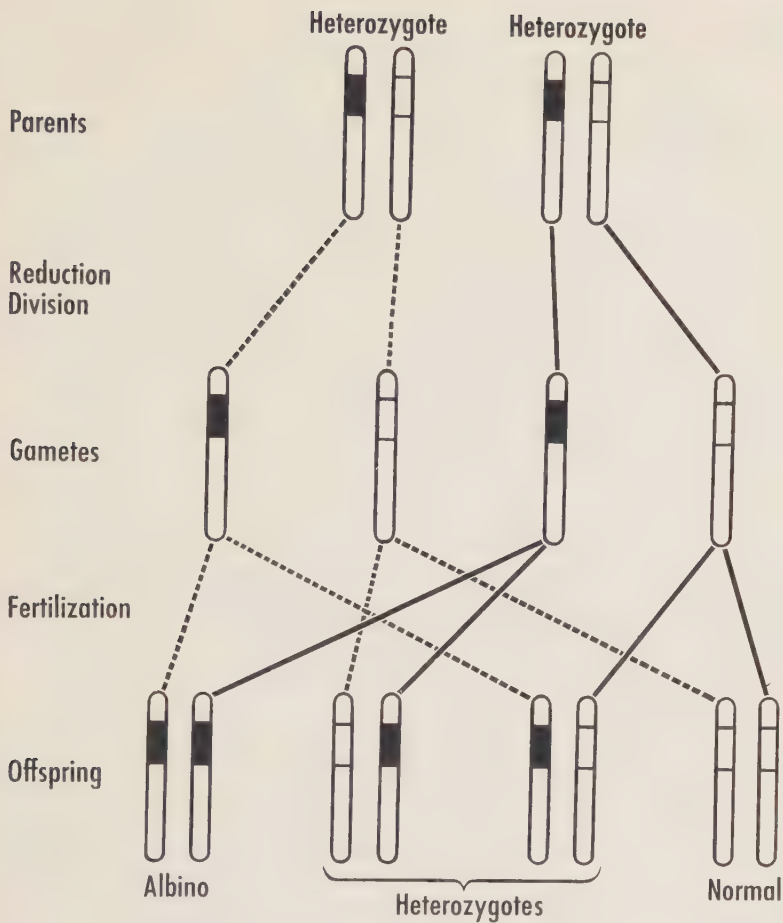


FIG. 28. The Recessive Form of Inheritance of Albinism.

Albino, because he will be homozygous for the recessive albino c gene. The mode of transmission of these genotypes is shown in figure 28.

Now supposing an albino cc marries a carrier Cc , what will the children be like? This has been worked out in figure 29, thus: First, draw a large square with nine smaller squares within it. Put one of the albino parent's c genes in the last topmost square,

FATHER MOTHER	SPERM	SPERM
	C	C
OVUM	NORMAL C c	NORMAL C c
OVUM	ALBINO C C	ALBINO C C

FIG. 29. An albino father homozygous for the recessive albino gene cc , mates with a woman who is heterozygous for the same gene Cc . Half the children are normal but carriers, and half are homozygous albinos.

and another c in the middle topmost square; let us say these are the sperms from the father who carries only c genes in all his sperms. The mother is a carrier of the type Cc , half her eggs having C and half c . Put C in the middle square of the first column of small squares, and c in the square under that. Now put C in the middle and last squares of the middle horizontal column of squares and bring down the c from the top squares and place them next to C ; you should have Cc in each of the two squares. Put small c in the middle and last squares of the lowest level of the diagram and bring down the small c in the topmost squares and put them with the other small c 's in the middle and the last of the lowermost row of squares. All this rigmarole simply amounts to saying: mate the first vertical column with the first horizontal column and put the offspring in the four empty squares, as in the diagram below. You will see that from such a mating of an albino with a heterozygote, half the children will be albino and half normal but carriers.

What happens when an albino marries a normal person who

does not carry the recessive gene? You should be able to work out the answer to that question for yourself. Such a marriage would involve a CC normal and a cc albino, since the gene C for normal pigment is present in *all* the germ cells of the homozygous parent and is a dominant, all the children of such a marriage will be normal but they will all be carriers of the recessive gene since they will be of the genotype Cc . Diagram this for yourself, using a diagram like the one shown in figure 29.

You can use this same type of diagram to work out the hereditary mechanism of many other conditions.

While in the majority of recorded cases albinism is transmitted by a recessive gene, in some cases the condition is transmitted by a dominant gene. Thus, we have in albinism an example of the same phenotype being conditioned by two different genotypes.

Skin Color in General

While the heterozygous parents of albinos appear in every way to be normal, they do, in fact, show one phenotypic evidence of the presence of the defective pigmentary gene, namely, the translucency of the iris to light. If you have a brunet complexion you will know that as a result of more or less prolonged exposure to sunlight your skin can turn many shades darker than it normally is. This change of color is due to an increase in the amount of melanin in your skin, which is activated by sunlight. If you are blond or fair-complexioned, you will know that you tend to burn more readily upon exposure to sunlight than your brunet acquaintances, and that you do not tend to tan as quickly as they do. This is due to the fact that fair-skinned people have much less melanin in their skin than brunets, and hence burn more easily and take longer to increase the melanin content of their skin. From the evolutionary standpoint this genotypic capacity of the melanin content of one's skin to change has been of great adaptive value in areas of varying sunlight intensity; allowing low-pigmented skins to permit the passage of enough sunlight for the manufacture of vitamin D in the body and, when the sunlight continues, to permit the skin to respond with the development of more melanin. Some brunets can turn virtually ebony-black upon prolonged exposure to sunlight; others don't get quite so dark. The same is true of fair-skinned people. It is, I think, fascinating

to observe how really dark a fair-complexioned person can get, the blond hair, blue eyes, and very dark skin making a most striking picture. The point, however, of these comments is to draw attention to the fact that all sorts of gradations in skin color can be achieved by the pigmentary system in response to sunlight. This suggests that skin color is the effect of different degrees of pigment intensity controlled by the action of many genes, that there are really no fundamental differences in the kinds of genes which are responsible for the different skin colors of different ethnic groups, but the range of color is merely a matter of the differences in the frequencies of the same kinds of genes. We have already learned that color genes retain their identity and segregate out unaltered in each generation, though in effect they appear to produce blending of pigments. Clearly the interaction of genes is involved, and judging from the whole range of colors of which human skin is capable, it is clear that many genes must be involved, each producing a small effect without dominance. Such genes are called *multiple genes* or *polygenes*. As I have already said, we do not at present know how many genes may be involved. It is probable that there are at least six pairs of genes at work.

If, for the sake of illustration, we assume that six pairs of genes are involved, and we consider these genes only in Negroes and in whites, then we can assign three pairs of heavy-pigment genes to Negroes, of the genotype $P_1 P_1, P_2 P_2, P_3 P_3$, and to whites three pairs of light-pigment genes $p_1 p_1, p_2 p_2, p_3 p_3$. The more P genes a person has the darker he will be, for the effects of the P genes are additive, and the more p genes a person has the lighter skinned he will be, for the light-skin genes are negatively additive, as it were. Now, if we say that the first pair of genes in the Negro series have the heaviest pigment, and the second pair a moderate amount of pigment, and the third pair the least heavy amount of pigment, and we say that the first pair of genes in the white series have a small amount of pigment, the second pair a smaller amount of pigment, and the last pair the smallest amount of pigment, remembering that in whites the amount of pigment is much less than in Negroes, it will be seen that on a six-gene basis involving three pairs of genes having different pigment strengths that you can get sixty-four or 2^6 possible gene combinations, yielding almost as many degrees or kinds of Negro and white skin color. Actually, we know that there are more variations than these, but the choice of number of gene pairs was arbitrary.

The offspring of Negro-white unions will be of the genotype $P_1p_1P_2p_2P_3p_3$; that is, the offspring have three dark-pigment genes and three light-pigment genes, so that they will be intermediate in color between both parents. The sex cells of mulattoes will contain three genes in eight different combinations, that is, in haploid number, as follows, $P_1P_2P_3$, $P_1P_2p_3$, $P_1p_2P_3$, $p_1P_2P_3$, $P_1p_2p_3$, $p_1P_2p_3$, $p_1p_2P_3$, and $p_1p_2p_3$. When mulattoes marry, their offspring, in any population in which there are many such marriages, are likely to show the following distribution of skin colors: about 20, or almost one third, of the F_2 generation out of every 64 individuals will carry three dark-pigment genes and three light-pigment genes, and will therefore resemble their parents in skin color. About 30, almost half, will carry two dark- and four light-pigment genes or four dark-pigment genes and two light-pigment genes, and will therefore have either somewhat lighter or somewhat darker skins than their mulatto parents. About 6 out of the 64 will have five dark- and one light-pigment genes, and will be intermediate between Negro and mulatto, and about 6 will have one dark-pigment gene and five light-pigment genes, and will be intermediate between mulatto and white. One out of 64 will have six dark-pigment genes and will be completely Negro in skin color, while another one will have six light-pigment genes and will be completely white in skin color. This is diagrammatically illustrated in figure 30.

It should be obvious that a white child cannot be born of the union of two Negroes, nor can a Negro child be born of the union of two whites. If the parents' skin is Negroid then they carry Negroid genes, and they can have only children with Negroid skin color. If the skin of the parents is white, then they carry only light-pigment genes and can therefore have only light-skinned children. If one of the parents carries one dark gene, that is to say, is of the genotype $p_1p_2P_3$, and marries a person who carries no dark genes and is of the genotype $p_1p_2p_3$, owing to the fact that the skin-color genes are neither dominant nor recessive the five light-pigment genes will almost completely "wash out" the effects of the dark-pigment gene in the "blending" of the pigmentary factors. The stories about "black" children suddenly being born to parents who were whites or who had some concealed Negro ancestry are complete nonsense. If the parents have white skins their children can have only white skins.

When mulattoes marry Negroes or whites the skin color of most

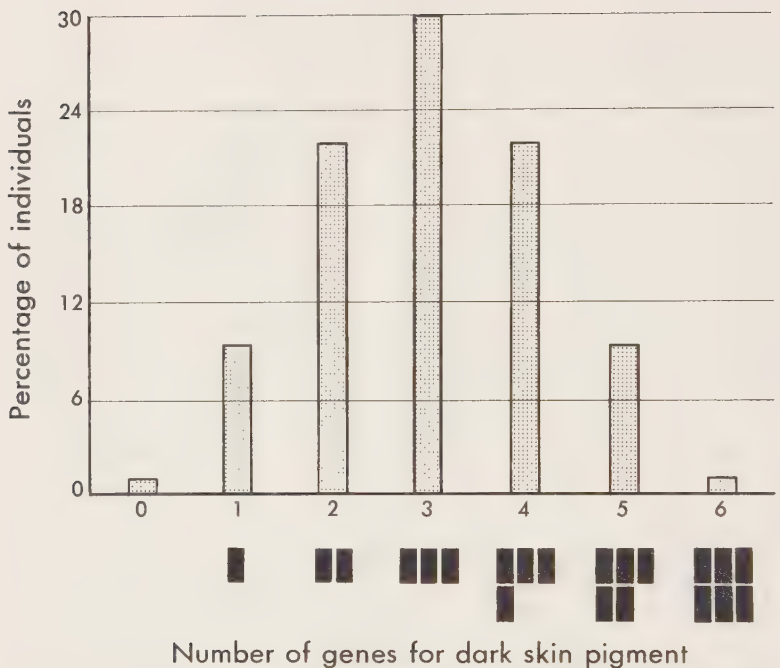


FIG. 30. Inheritance of Skin Color in Negro-White Mating, assuming three pairs of pigment genes without dominance. The heights of the columns show the percentages among the second generation hybrids of individuals with no color-producing genes, with one, two, three, four, five, and six. (After Dobzhansky, *Evolution, Genetics, and Man*.)

of their children is intermediate between that of the parents. Three eighths of the sex cells of mulattoes carry two dark-pigment genes, three eighths one dark-pigment gene, one eighth three dark-pigment genes, and one eighth no dark-pigment genes. Since a Negroid sex cell carries three dark-pigment genes, the offspring of a mulatto-Negro cross would yield three eighths with five dark-pigment genes, three eighths with four, one eighth with six, and one eighth with three dark-pigment genes. A mulatto-white offspring would yield three eighths with two dark-pigment genes, three eighths with one, one eighth with three, and one eighth with no dark-pigment genes.

The Sacral Spot

The sacral spot, sometimes called "the blue spot," or "the Mongoloid spot," is a largish, bluish area found at the base of the spine over the area of the sacrum and adjoining buttocks. It is found in about 90 per cent of Negro infants, in many Mongoloid infants, and in some American Indians and some dark-complexioned whites. It usually disappears within six months to a few years. It is never seen in blond children with blue eyes.

Finger Smudges

Some infants with some Negroid ancestry show "smudges" of pigment on the backs of the fingers between the joints. When present these indicate that the child will develop a dark complexion. When absent the indication is that the skin color will develop as white.

Freckles

The small yellowish-brown-pigmented spots seen on the face and other parts of the body apparently behave as dominants in heredity. One or the other parent is usually also freckled. Freckles in some individuals only become evident after they have been exposed to sunlight. In all individuals the intensity of the pigment in them increases following appreciable exposure to sunlight. That is why freckles tend to be reduced in winter and to blossom in summer. Freckles appear to be more frequent in persons with red or a reddish shade of hair. It is interesting to note that many chimpanzees have freckled faces.

The Skin of Redheads

The skin of redheads is probably lacking in certain pigmentary factors, and this appears to be in some way connected with the inadequacies of their blood-clotting capacities. This is a subject about which we know practically nothing and on which we need much systematic observation. There is some evidence that the bleeding time of redheads is longer than that for brunets or

blonds. Some obstetricians take special precautions when a redhead is to be delivered of a baby because of the tendency of some redheads to bleed profusely following delivery. The skin of redheads is very much more susceptible to burning by exposure to sunlight than is that of brunets or blonds. Red hair behaves as a recessive in inheritance, and so apparently does the gene for the character of the skin that is almost invariably associated with red hair.

That red hair, as distinct from Titian red, has some profound constitutional ramifications is suggested by the fact that tuberculosis in redheads generally runs a much more severe course than in non-redheads. Interestingly enough, the same seems to be true of individuals inclined to freckling. There is also some evidence that redheads have greater difficulties with anesthetics.

Eye Color

The color of the eyes is determined by the presence in the iris of the very same pigment, melanin, that is so closely related to the color of the skin. The *iris* (Greek, rainbow) of the eye has received its name from the rainbowlike variety of colors it exhibits in different individuals. The iris is a circular, disklike structure, the central opening of which is the pupil of the eye. The iris acts as a diaphragm controlling the amount of light admitted to the eye. The amount of pigment in the iris also serves a similar function. Hence, it is found that in high-sunlight areas where skins are deeply pigmented the iris is too. In low-sunlight areas there is less pigment in the iris. The color of the eye is dependent upon the amount of pigment present in the front and back portions of the iris. The pigment is contained in fine particles which vary in size and shape.

The iris has a double layer of pigment cells at its back, and a front layer of cells that may or may not be pigmented. The color of the eyes is conditioned by two principal factors: (1) the amount of pigment in the back layers of the iris, and (2) the amount of pigment in the front layer of the iris.

If the pigment is present only in the back portion of the iris, the eyes are blue.

If the pigment is also present in the front part of the iris in a moderate amount, the eyes are gray or green.

If the pigment is abundant in both parts, the eyes are brown or black.

The thickness of the iris also modifies eye color.

There are no such things as blue or green pigments in the iris: the different eye colors are due to the different reflective properties of the brown pigment particles, as determined by their density, distribution, or scattering, and the thickness of the iris. The blueness of the eye comes about in much the same way as the blueness of the sky, which is caused by the reflection and dispersion of light from the particles of dust and other substances in the air. The eyes of all newborn babies of every human variety are blue. The reason for this is that, whatever their genetic eye color (which will later become apparent), the pigment particles do not develop in the front part of the iris till some time after birth.

There would appear to be at least one pair of genes, B, b , involved in eye color, yielding the genotypes BB , Bb , and bb , B being incompletely dominant so that dark brown would always be BB , light brown Bb , and blue bb . In each of these three genotypes there are many variations not only in shade but also in the distribution of several colors in the same iris, as green, brown, yellow, and violet in one and the same iris. Also the two irises in the same individual may differ in color. Clearly many more than the pair of major allelic genes are involved to produce these varying eye-color effects; such gene pairs are called *modifiers*. Thus, two blue-eyed parents who are each of the genotype bb would give birth to children who were exclusively of the same genotype, but some of these children might in fact have sufficient brown pigment in their irises to cause them to be classified as brown-eyed, and this would normally be due to the action of modifying genes. Metabolic changes during embryonic development conceivably may play a role in some cases.

In albinos, owing to the failure of the general development of pigment throughout the body, the eyes appear pink—the pinkish color being due to the redness of the small blood vessels showing through the iris. It is interesting that the gene for albinism in rats, mice, and rabbits also affects the disposition of these animals. The albino animals are much more gentle, mild, and curious than even their own pigmented litter-mates. There is some slight evidence that something of the same sort may also occur in man.

Color Blindness

This has already been dealt with on pages 180–183.

Hair Color

A hair is essentially a hollow cylinder the central canal (medulla) of which is filled with pigment particles, air, and oils, while the outer shaft (cortex) contains pigment granules in dark hair, and air in white hair. A single layer of flat scales (the cuticle) overlies the cortex. It is the interaction of all these parts and their contents which, in the presence of light, is responsible for the color of the hair. It is well known that under different varieties of light the color of the hair will appear correspondingly different. It is also known that under different environmental conditions the hair may vary somewhat in color; for example, light brown hair will often turn to blond during the summer as a result of the bleaching effect of the sun. During illness the hair may often lose its glossiness. But there is no evidence whatever, that will withstand a moment's critical examination, that hair is capable of turning gray overnight. None of the claims that have been made have withstood such critical examination.* It is extremely unlikely that hair could naturally change in color overnight, for the simple reason that the visible hair is a dead structure which contains inert pigment, and it is quite impossible to see how this could be naturally affected. When hair starts turning gray it is observed that it does so first at the root, while the terminal portion retains its color, so that we have a hair that is gray at the base and pigmented toward the end.

Hair color ranges all the way from the pigmentless straw color of the albino to the deep black to be found among all the peoples of the earth. The number of different hair colors in the human species must be very large indeed. No one has yet made a complete census of them. There is perhaps no other single mammalian species that presents so large a variety of hair colors as man.

Like skin color the genetics of hair color is not, at the present

* For an excellent discussion of this interesting subject see Bergen Evans, *The Natural History of Nonsense*.

time, clearly understood. A number of different genes are undoubtedly involved. Dark hair appears to be dominant over blond hair. Dark-haired parents may have children with almost any variety of hair color. Blond-haired parents always have blond-haired children and never have dark-haired children, though they may have red-haired children. These facts indicate that blond hair is recessive to dark hair, so that individuals with dark hair carry either two dark-hair genes or one dark-hair gene and one for another color while blond individuals carry two blond genes.

Red hair seems to be due to a distinct gene, and this usually behaves like the genes for blond hair, usually being recessive to the darker hair colors, brown and black. Dark-haired parents often produce a redhead. Hence, individuals with red hair either carry one or two genes for redheadedness. The offspring of two redheads will generally, but not always, be redheads. The people who produce the largest number of redheads are the highlanders of Scotland, 11 percent of whom exhibit the trait. In the cases in which only one gene for red hair is present, whether the hair of the individual will be red depends upon the "strength" of that gene in relation to its opposite member (its allele). If the gene for red hair is stronger than its opposite member or members, say for dark or blond hair, it will express itself as a function both of its own "strength" and that of the gene or genes for other hair colors. It is for this reason that we often see individuals with different shades of red hair, sometimes with red and brown hair on the same head (to be carefully distinguished from the variety that comes out of a bottle).

The genetics of hair color is complicated by the fact that the hair contains at least two pigments. One of these pigments, a granular melanin, varies in intensity from a light, golden blond through shades of sepia to black. The second of the pigments varies qualitatively from yellow to dark red. Hair color is therefore a result of the intensity and the quality of pigment. There are genes that affect intensity, as illustrated by dark hair in relation to light, and genes that affect quality of pigment, as dark hair in relation to red. The fact that there are so many different shades of hair indicates that melanin pigment is dependent upon the action of several genes. Not only this, but these same genes appear to act differently with age. This is seen in many individuals who are blond-haired in childhood and become dark-haired as they grow

older. Among the Australian aborigines, who all have dark brown hair as adults, children are sometimes born who are fair-haired and who remain so until they approach puberty, when their hair invariably darkens and gradually assumes the dark brown color of the adult. Melanin production, in such individuals, apparently increases from childhood to maturity. To some extent this also appears to be true of skin color, although the changes in the darkening of the skin appear to proceed much more rapidly than in the case of hair color. The darkening of the hair with the advent of puberty appears to be the result of the action of the increase in the quantity of the sex hormones. The pigmentless hair of albinos is due to the pairing of recessive genes for hair color which lack all pigment-making capacity.

Partial albinism of the hair occurs in some individuals in the form of a white forelock. This is usually limited to the area of the hairline where it meets the forehead. When it is allowed to grow long and is combed backward, it gives the false appearance of a large tract of hair being involved. The condition is inherited as a simple dominant. It is a contemporary fashion among some females to dye the hair in imitation of such a forelock, usually in some blondish color. Partial albinism of the hair is generally associated with some form of partial albinism of the skin, if only a spotting of an extremity or of some other part of the body.

Graying of the hair is also dependent upon the action of a single dominant gene sometimes affected by modifying genes. Graying in identical twins will usually occur at precisely the same time and in the same pattern. The age at which the hair turns gray is inherited as a dominant, as is the tendency to graying of the hair. In some individuals the hair never turns gray even in extreme old age. I know of no investigations on this subject, but from my own random observation it seems likely that the condition of non-graying is inherited as a dominant. Blondes are said to have more hair on their scalps than dark-haired individuals, and redheads are said to have the least.

Hairlessness

There are several different kinds of hairlessness (hypotrichosis), ranging from the type which is congenital to that which is induced by disease or disorder. Conditions of hairlessness which in the one

type are due to recessive and in the other type due to dominant genes have been reported in man. These conditions are not to be confused with baldness which affects only the hair of the scalp, whereas general hairlessness may affect every part of the body.

Baldness

Baldness has already been discussed on pp. 186–191 as an example of a sex-limited trait.

Eyebrows

Eyebrows may vary in every conceivable way. They may be thick, thin, scanty, cross over the nose, be arched, or straight. The hair usually points away from the nose, but in many instances the hairs lying nearer the nose point in its direction or upward. Such traits are all inherited, and, save thin and scanty eyebrows, appear to behave as dominants.

Eyelashes

The character of the eyelashes, their thickness, length, and curvature are all inherited traits, and again appear to behave as dominants in the case of long lashes, and recessive in the case of short ones. If the parents are homozygous for long lashes all the children will have long lashes, but if the parents are heterozygous for these traits some children will have long and some will have short lashes.

An interesting thing about eyelashes is that their color at birth is a good indication of the color that the body hair will assume in its final form—excluding gray.

Face

Mustache and beard hair are under the influence of separate genes, at least with respect to color. In some men the beard may be black or dark brown, but the mustache may be blond or red. The difference in coloration may remain throughout life or until graying sets in. The distribution of the hair on the face, its color, and form are all genetically determined and are traits which ap-

pear to behave as dominants. The growth of facial hair in men is largely controlled by the male sex hormones, the absence of these hormones being responsible for the lack of growth and development of facial hair in the female.

Hypertrichosis of the Ears

The dense growth of hair on the outer ears, known as auricular hypertrichosis, occurs only in men and until recently was believed to be sex-linked to the Y chromosome and was therefore thought to be transmitted directly only from father to son. The gene responsible appears to be sex limited, for the author knows of a case of two brothers where one exhibits the condition while the other does not. Something of the difficulty of genetic investigation of man is illustrated by the fact that although the author was very friendly with the hypertrichotic brother, when inquiry was made as to whether he would be agreeable to a study of the condition in his family, he promptly and firmly declined with the words: "Why should I be a guinea pig!" Fortunately, most people react in a much more co-operative manner to such inquiries.

Whorls and Cowlicks

Some males have a difficult time keeping the hair near the back of their crown from standing up like a clump of untidy hay. This is the hair of the hair whorl. It emerges near the top of the back of the head in a turn usually to the right in a clockwise direction. The clockwise whorl occurs in the majority of individuals and is inherited as a dominant trait. The counterclockwise whorl found in some individuals is inherited as a recessive. The clockwise or counterclockwise direction of the hair determines the side on which the hair will be parted. Individuals with clockwise whorls part their hair on the left; those with counterclockwise whorls part their hair on the right. Parents with counterclockwise whorls will transmit them to all their children. Parents with clockwise whorls will transmit the clockwise whorl to all their children if they are homozygous for the dominant gene, or they will have some children with a counterclockwise whorl if they are both heterozygous for the counterclockwise whorl gene.

Double whorls occur and are inherited as a recessive trait. They may both go in the same direction or in opposite directions in different individuals. More rarely triple whorls are observed. These, also, are inherited as a recessive trait.

Cowlicks are misplaced whorls. They may occur on the forehead, the extremities, the back, the chest, or on other parts of the body. In most cases they appear to be inherited in much the same way as the crown whorl.

Body Hair

The growth, development, and distribution of the body hair varies in the two sexes. Body hair is generally much more profusely developed and distributed in the male than in the female and for the same hormonal reasons that influence the growth and development of the facial hair in the sexes. The pattern of growth and distribution of the body hair in both sexes is probably inherited in much the same manner as scalp hair.

Hands and Fingers

The development of hair on the backs of the hands and fingers is also under genetic control. A good growth of hair on the back of the hand is inherited as a dominant trait, the absence of such a growth is inherited as a recessive. Similarly, the presence of some hair on the middle segment of the middle, ring, and little fingers is inherited as a dominant trait, while the absence of such hairs on the middle segments is inherited as a recessive trait. It is extremely rare for hair to be present on the middle segment of the index finger. Why this should be so is a mystery. It is believed that at least five genes are directly involved in influencing the presence or absence of mid-digital hair.

Hair Form

Hair form is usually classified as straight, wavy, curly, kinky, and coiled (peppercorn tufts, as among the Bushmen of South Africa). When both parents have straight hair the children are all usually straight-haired. Wavy- or curly-haired parents if they are homozygous for waviness or curliness will have only wavy- or

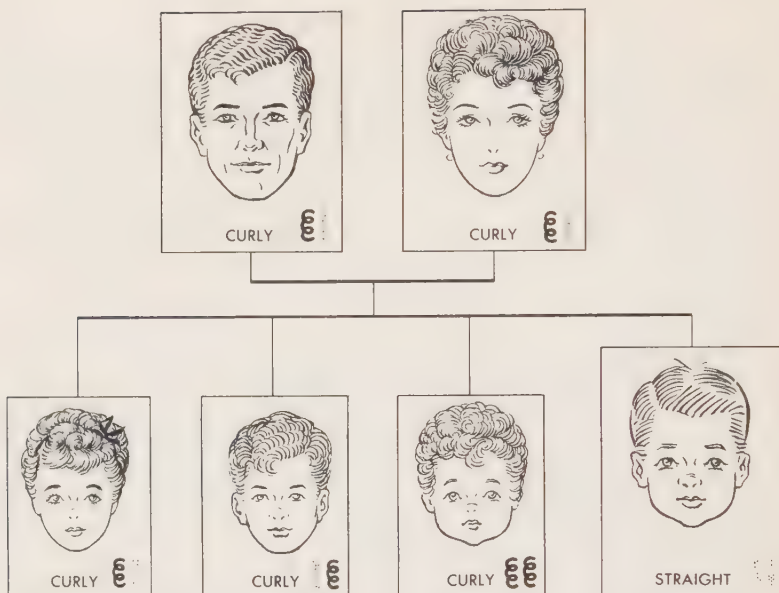


FIG. 31. Inheritance of Hair Form. The symbols stand respectively for curly and straight hair. Curly dominates straight. Here we see the parents heterozygous for curly and straight hair yielding a 3 curly to 1 straight hair ratio in their four offspring.

curly-haired offspring, but if they are heterozygous for these traits, some of their children will be straight-haired, some curly-haired, and some wavy-haired. Of course, each of these types of hair is variable in its character. Wavy hair may be characterized by low waves, medium waves, and deep waves, and the same applies to curly hair. Straight hair may be coarse, thick, or thin, and the number of hairs per square centimeter may vary from person to person. The peoples of the Mongoloid major group of mankind have fewer hairs per square centimeter on the body than do members of the other major groups of mankind.

When a kinky-haired person marries a white, their offspring's hair is kinky; this fact establishes the dominance of kinky hair over all other forms of hair. When a curly-haired individual marries a wavy-haired person, the tendency is for the curly-hair gene

to dominate over the wavy-hair gene. The wavy-hair gene tends to dominate over the straight-hair gene. Hence, it seems as if the greater the quality of curliness the higher the quantity or degree of dominance.

While kinky or woolly hair is a normal characteristic of Neogroids, such hair has appeared as a mutation in whites of exclusively white ancestry. Three Norwegian families in which the mutation appeared have been described by Professor O. L. Mohr of the University of Oslo, and the author of this book has observed the same condition in an American white female of exclusively white ancestry.

The Adaptive Value of Hair

A feature so widely distributed over the body and so highly differentiated in the various parts of it as the hair in man is almost certainly of considerable adaptive value. The adaptive value of a trait is the extent to which it improves the chance of organisms, in the environments they inhabit, of survival and of leaving progeny, as compared with those of their kind not possessing the trait. What, then, is the adaptive value of the different types of hair on the body and its peculiar distribution? And what is the value of the different forms of hair and their distribution over the various parts of the body in the different major and ethnic groups of man?

These are questions to which the answers can only be conjectural, and our conjectures can only be evaluated in terms of a high or low degree of probability.

In the first place, hair is, as it were, a continuation of the nervous system. It is derived from the same embryonic layer as the nervous system, namely, the ectoderm. * Among its most impor-

* The three primary embryonic layers of cells from which the various organ systems of the organism are developed are the endoderm, the mesoderm, and the ectoderm. The innermost layer, the *endoderm*, gives rise to the linings of the digestive and respiratory tracts, of the digestive glands, of the bladder and urethra, and of the thyroid and thymus glands. The middle layer, the *mesoderm*, gives rise to muscles, bones, cartilage, the dentine of the teeth, ligaments, kidneys, ureters, ovaries, testes, heart, blood, the vessels which carry the blood and lymph, the external covering of the heart, and the linings of the chest and abdomen. The *ectoderm*, the outermost layer, gives rise to skin, hair, nails, oil glands, lining membranes of the nose and mouth, the salivary and mucous glands of the nose and mouth, and the nervous system.

tant functions is the transmission of tactile sensations from the outside world. The sensory disturbance does not occur in the hair itself, for this has no nervous supply of any kind, but pressure of any kind on the hair is felt at the base of the hair follicle and the adjacent skin, which *is* supplied with sensory nerves, and it is through these that disturbances on the surface of the hairs are transmitted to the brain and spinal cord. Hair, therefore, among its many other functions, constitutes a sentry, as it were, standing guard on the outer ramparts of the body, announcing the arrival of visitors of whom one might not otherwise be aware. Civilized people who habitually wear clothes are not as sensitive to such disturbances as are nonliterate peoples who habitually go unclothed. Individuals living in nonliterate societies, though less hirsute as a rule than those living in civilized societies, are likely to be aware of the presence of an insect on their bodies long before the white becomes conscious of it. The adaptive value of hair on the unclothed under primitive conditions as a protection against the unsolicited attacks of various noxious insects should be obvious, for tactile hairs are extremely important in apprising us of the advent upon our bodies of objects long before those objects reach our skins.

Hair in the armpits reduces the friction as the arm moves upon the body, and hair upon the pubic regions of the sexes may serve a similar function when two bodies are involved, as in coitus.

Head hair has a protective value in many ways: (1) It serves to protect the head from the elements, for example, against (a) excessive sunlight's penetrating rays, (b) heat, (c) cold; (2) it serves to maintain the normal temperature of the brain. The black kinky hair of the Negro is most efficient in these respects under conditions of tropical sunlight. The hair directly absorbs most of the sunlight, acting as a sort of air chamber, while the sunlight which does penetrate beneath the skin activates the scalp sweat glands. The sweat which is then secreted forms a wet blanket. The combination of air chamber and wet blanket effectively serves to protect the underlying tissues from damage from excessive light and heat.

The eyebrows may function to prevent sweat that may be running down the forehead from entering the eyes. The eyebrows may also serve to reduce some of the light entering the eyes by both absorbing some of it and also acting as a shade.

The function of eyelashes is apparently manifold. Eyelashes serve to protect the eyes by preventing particles from entering the eye, as hairs in the nostrils do, by acting as tactile sensory warning mechanisms, and by absorbing light (persons without eyelashes are forced to squint).

Why men have beards and mustaches and women don't is a mystery. If, as has been suggested by some writers, the beard serves to protect men against freezing temperatures, then why doesn't the biologically more valuable part of the species, woman, have a beard, too? The fact is that in freezing temperatures a beard is a disadvantage, for when the condensed water from one's breath freezes on one's beard, the skin beneath freezes all the more readily. Arctic peoples have very little hair on the face, and what little they may have they either pluck or shave. Mongoloid and Negroid peoples have very little beard or body hair. If the beard owes its origin to sexual selection rather than to natural selection, why are Mongoloid and Negroid men so sparsely endowed with these appendages? So the mystery remains. Perhaps baldness and beards are the purely fortuitous penalties that men must pay for being so constantly perfused with androgens.

The Features

The Nose

THE NOSE IS a very complex structure made up of many different parts, so that many genes must be involved in its construction. Yet, as everyone knows the power of heredity is often strikingly exhibited in this unique projection—for among the Primates, the Order of Mammals to which man belongs, there is no other member who possesses such a bony cartilaginous prominence. One monkey, the Bornean *Rhinopithecus nasalis*, possesses a remarkable schnozzle, but this lacks the bony eminence of man's nasal bones and is mainly cartilage and soft tissue. Man's nose is largely due to the shortening of the projection of his jaws which, as it were, has left this peninsula of bone and cartilage projecting into space.

In spite of the fact that different genes are involved in the formation of the different parts of the nose, the form of the nose may be inherited as a whole, which would suggest that the genes responsible for nose form are situated closely together on the chromosome. But this is highly probably not the case. Independent features of the nose are quite as often transmitted as such and not as part of the whole complex of genes derived from a single parent. Because these features are capable of being inherited independently, we shall understand the mechanics of the heredity of nose form much better if we study the individual features of the nose.

THE ROOT. The root of the nose is that part which joins the forehead, and is often miscalled the bridge. The bridge emerges from the root and corresponds to the anterior bony projection of the nasal bones and the cartilage which joins it. It is the

bridge of the nose which runs into the tip below. The root of the nose may be flat as in Mongoloids and to a lesser extent in Negroids, or it may be of medium shallowness, or high as in the so-called Greek nose in which the forehead seems to run without any break directly into the nose. The high root appears to be dominant over the medium root, while the flat or concave root would appear to be recessive.

THE BRIDGE. The angle at which the nasal bones emerge from the root largely determines the degree of projection of the bridge. Hence, both genes devoted to the root and genes devoted to the projection of the nasal bones participate in giving the bridge its particular form. If one inherits flat or low nose-root genes from one parent and high bridge genes from the other, a projecting or concave bridge may result. If the root is elevated, the bridge of the nose will tend to be straight. If the root is high a prominent convex or Roman nose may result, and if the root is very high a Greek straight nose may result. The genes for the prominent convex Roman nose are dominant over the genes for the straight nose. The genes for the high and narrow bridge dominate those for the low and broad bridge.

THE TIP. An amazing variety of nose tips are encountered from sharp and depending to bulbous and upturned. Some tips exhibit a middle furrow. Round and square tips are encountered in whole families, as are *retroussé* or upturned ones.

THE NOSTRILS. It is not known whether the shape of the nostrils is inherited as an independent trait or whether it is dependent upon the form of the nose. The latter explanation would seem to be the most probable, for there is a definite correlation between the form of the tip of the nose and the wings and nostril shape, small noses with round tips being characterized by small and pear-shaped nostrils, while high bridged noses with square tips have long and slitlike nostrils. Nearly circular nostrils go with bulbous-tipped and concave noses. Such evidence as there is indicates that broad nostrils are dominant over narrow.

THE WINGS OR ALAE. The wings of the nose are situated on either side of the nostrils, the nasal septum separating one nostril from the other. The wings may be flaring (popularly associated with hot-tempered individuals—an alleged association which has, however, never been scientifically investigated), or they may be more or less closely approximated toward the septum. They may

be cut high, so as to expose the septum, or they may be situated at a lower level than the septum.

The Upper and Lower Jaws

The front teeth of the upper jaw normally bite over the front teeth of the lower jaw, but in some individuals the lower jaw is longer than the upper jaw and the upper front teeth bite behind the lower front teeth. Sometimes, as in the case of the royal house of the Spanish Hapsburgs, in whom the condition has been traced back for more than six hundred years, a protruding lip is associated with this type of undershot jaw. Both conditions are due to independent dominant genes.

THE CHIN. The chin is unique to man; no monkey or ape possesses a protuberant front of the lower portion of the lower jaw, and no man possesses the kind of receding jaw that apes and monkeys do. But some human jaws give the appearance of being more receding than others, while some are definitely more projecting in the chin region than others. There is a common belief that a prominent chin denotes strength of character, but for this belief there is not the slightest scientific evidence.

When the chin protrudes beyond the plane of the face, it is described as prominent. When the chin is in the same plane as the face it is described as normal, and when it is situated behind the plane of the face it is described as receding. The receding chin is believed to be caused by a recessive gene, the normal chin by a dominant gene, and the prominent chin by a dominant gene. A narrow chin appears to be recessive to a wide chin, and a long chin, of more than two inches from the level of the mouth, appears to be dominant to a short chin.

THE TEETH. Malocclusion of the teeth—that is to say, disordered relations in their biting surfaces—crowding, rotation, reduction in size or number, failure to erupt, impaction, even caries, are usually the consequences of genetic factors. Malocclusion of the teeth can usually be corrected, but in some cases the genetic factor is so strong that even when the occlusion has been put into good shape the teeth will return to their former relations. I have known one such case to occur not less than three times—in the case of a dental nurse! Malocclusion appears to be due to a dominant gene which is sometimes incomplete or irregular

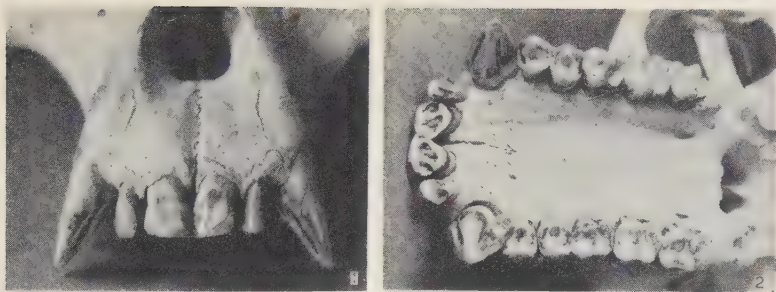


FIG. 32. Premaxillary Diastema in the Gorilla. 1. Frontal view. 2. Palatine view.

in its expression. The other conditions named also appear to be due to a dominant gene of a similar nature. There is good evidence that the development of the different parts of each tooth is dependent upon many genes. For example, a tooth may develop perfectly except for complete absence of enamel or dentine or some other structure of the tooth. A search of the individual's pedigree will often indicate similar conditions, thus suggesting that each of these structures of the tooth is genetically conditioned.

It is not possible here to deal with the twenty teeth of the deciduous (shedding or milk) dentition or with all the thirty-two teeth of the permanent dentition. I shall therefore restrict myself to the tooth that is most frequently genetically affected, namely, the upper lateral incisor.

If you will look at the upper jaw of any monkey or ape you will see that between the canine (eye) tooth, and the lateral incisor there is a diastema (space) into which the projecting portion of the lower canine tooth fits. The bone which affords this space is the premaxilla. In the course of evolution man's teeth underwent a reduction in size, especially the canines (though the tips of which still project beyond the occlusal or biting level of the other teeth). This reduction of the canines rendered the premaxillary diastema quite unnecessary, and so it eventually disappeared. But in disappearing, the area involved has become somewhat unstable. Because this region of the jaw involves the development, meeting, and subsequent fusion of two separate bones, the maxilla and premaxilla, all within a period from the

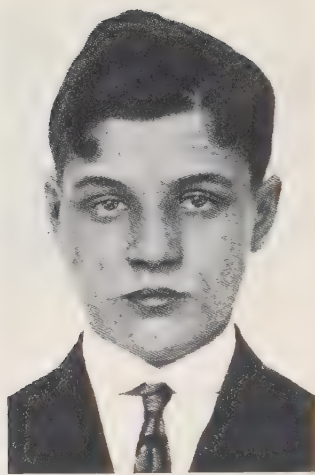
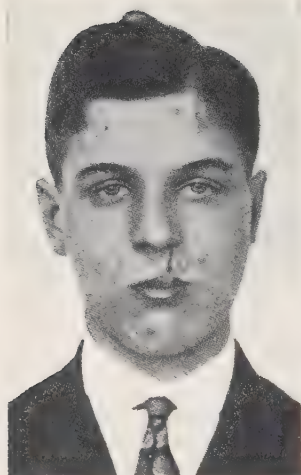


FIG. 33. Identical male twins, one born with hare lip and cleft palate, and the other perfectly normal. (After von Verschauer.)

end of the seventh week to the end of the ninth week of embryonic development, the premaxillary area (in front of the eye-tooth on each side) appears to be easily disordered, in some ethnic groups more than in others, with the result that anomalies of development all the way from an undescended lateral incisor to its complete absence or cleft palate and harelip may result.

Among modern whites the upper lateral incisors are missing in about 2.5 percent of the population. They are markedly reduced in size in about the same percentage of cases, are slightly reduced in about 17 percent, rotated in about 4 percent, crowded in about 7.5 percent, and in about 4 out of every 1,000 cases they are duplicated. It is interesting that the Chinese and Japanese exhibit a higher frequency of degenerate lateral incisors (7 and 4.7 percent respectively) but fewer missing lateral incisors (0.15 and 1.1 percent) than whites. Missing lateral incisors are rare among African Negroes, but occur in over 2 percent of mixed American Negroes. It is extremely rare to find missing lateral incisors among the native peoples of the Pacific such as Polyynesians, Melanesians, and Australian aborigines, so that in these peoples this region of the upper jaw and teeth is not an area of instability. Anomalies of the lateral incisor are slightly more frequent in females than in males.

CARIES. Caries or tooth decay is known to have a familial incidence. In 1946 Klein published a study of 5,400 individuals, in which he found that the highest incidence of decayed, missing, and filled teeth (DMF experience) occurred in individuals whose parents also had high DMF rates; the individuals with a moderate DMF experience had parents with a similar moderate DMF experience, while those with a low DMF rate had parents with a low DMF experience. When the DMF rate of the mother was low, differences in that of the father were closely related to the rate of the sons but only slightly to that of the daughters. When the DMF rate of the father was low, differences in the DMF rate of the mother were closely related to the rates of both sons and daughters. It was concluded that there exist strong familial bases influencing DMF experience that probably have a genetic basis.

More recently, in 1958, Horowitz, Osborne, and DeGeorge reported on a series of forty-nine like-sexed pairs of white twins drawn from middle-income residents of New York City and ranging in age from eighteen to fifty-five years. Some of these pairs were one-egg twins and others were two-egg twins. It was found that the one-egg twins showed significantly more similarity in their caries experience than did the two-egg twins, thus strengthening the conclusion arrived at in 1940 by Nehls in Germany in a similar study that a hereditary factor exists for susceptibility to caries.

Early decay of the teeth has been recorded in several families in three succeeding generations, the incidence being consistent with a dominant gene as the principal factor. In one family the eleven individuals in three generations who were so affected were all females; the only three males in the pedigree had sound teeth. In this family a dominant sex-linked gene might have been involved.

Early decay limited to a single tooth has also been recorded in several generations. Resistance to decay also appears to be markedly influenced by genetic factors.

Harelip and Cleft Palate

Harelip and/or cleft palate occurs in about 1 out of every 770 births and is about twice as frequent in males as in females. The genetics of the condition in man is quite complicated and has not yet been fully worked out, but there is some reason to believe



FIG. 34. Ability to roll the tongue is inherited as a dominant. (Courtesy of A. M. Winchester, from *Genetics*.)

that the gene for harelip behaves as a recessive and that for cleft palate as a dominant with a variable degree of penetrance. Each condition may be inherited separately or together. There is good evidence that the development of the palate and adjacent structures is extremely sensitive to changes in the prenatal environment, and that severe emotional stress in the mother is capable of producing significant changes in this region (see page 102). Support for this view is furthered by the fact that where one identical twin has harelip or cleft palate or both, in about one fourth of the cases the other twin shows no signs of the condition. Harelip and cleft palate may be variously inherited either as a recessive, a dominant, or a sex-linked dominant. If the parents carry the genes for harelip and/or cleft palate in the recessive state, the chances are about 1 in 10 that they will have a child with a harelip and/or cleft palate. If one parent exhibits the condition, there is about a 2 percent chance of having an affected child.

The Tongue

Ability to roll the tongue, that is, to bring the sides of the tongue over the top, is inherited as a simple dominant. About 65 percent of people can do it. The inability to roll the tongue is inherited as a recessive.

TABLE XV
ABILITY TO TASTE PHENYLTHIOCARBAMIDE IN HUMAN POPULATIONS

Population	Place	Investigator	Number Tested	Per cent Tasters
Welsh	Five different towns	Boyd & Boyd (1937)	237	58.7
Eskimo, Unmixed	Labrador & Baffin Id.	Sewall (1939)	130	59.2
Germans		Gottschick (1937)	183	62.3
Danish	Copenhagen	Hartmann (1939)	596	62.8
Russians	Zagorsk (n. Moscow)	Boyd & Boyd (1937)	486	63.2
Arabs	Syria (interior)	Hudson & Peter (1934)	400	63.5
Russians	Kharkov	Boyd & Boyd (1937)	161	64.6
American Whites	Montana	Matson (1938)	291	64.6
Yemenites	Yemen, Palestine	Yunovitch (1934)	59	67.7
Armenians	Syria	Berberian (1934)	294	68.0
Ashkenazic Jews	Palestine	Yunovitch (1934)	245	68.5
American Whites	Washington, D. C.	Parr (1934)	439	69.1
Eskimo, Mixed	Labrador & Baffin Id.	Sewall (1939)	49	69.4
American Whites	New York and vicinity	Blakeslee (1932/35)	400	70.0
American Whites	Ohio State University	Snyder (1932)	3,643	70.2
Swiss	Zurich and vicinity	Botsztejn (1942)	544	70.4
Scottish	Glasgow	Riddell & Wybar (1944)	60	71.7
Irish	Dublin	Boyd & Boyd (1937)	398	71.8
Sephardic Jews	Palestine	Yunovitch (1934)	175	72.0
Copts	Cairo, Egypt	Boyd & Boyd (1937)	110	73.6
English	London	Falconer & Fisher (1947)	629	73.7
East Georgians	Tiflis	Boyd & Boyd (1937)	121	74.4
Basques	San Sebastian	Boyd & Boyd (1937)	98	74.5
Egyptians	Cairo	Hickman & Marcos (1934)	208	75.9
American Negroes	Alabama	Howard & Campbell (1934)	533	76.5
West Georgians	Tiflis	Boyd & Boyd (1937)	218	78.0
Mohammedans	Cairo	Boyd & Boyd (1937)	459	78.9
Flathead Indians, Mixed	Montana	Matson (1938)	442	82.6
Mixed Amerindians	Lawrence, Kansas	Levine & Anderson (1932)	110	87.2
Formosans, Chinese origin	Formosa	Rikimaru (1936)	5,933	89.5
Flathead Indians, Unmixed	Montana	Matson (1938)	30	90.0
American Negroes	Ohio	Lee (1934)	3,156	90.8
African Negroes	Kenya, East Africa	Lee (1934)	110	91.9
Amerindians	Alberta	Matson (1938)	310	92.4
Japanese	Japan	Rikimaru (1936)	8,824	92.9
Unmixed Amerindians	Lawrence, Kansas	Levine & Anderson (1932)	183	93.9
Chinese	Washington & New York	Chen & Chain (1934)	167	94.0
Formosans	Formosa	Rikimaru (1936)	1,756	94.8
African Negroes, Shilluk	Sudan	Lee (1934)	805	95.8
Amerindians	Northern Alberta	Matson (1940)	559	96.9
Navaho Indians	Ramah, New Mexico	Boyd & Boyd (1949)	269	98.2

Adapted from Montagu, A., *An Introduction to Physical Anthropology*.

The ability to taste various substances is also inherited. This fact was discovered when a chemist found the taste of the synthetic chemical substance phenylthiocarbamide (P.T.C.) somewhat bitter whereas a colleague of his couldn't taste it at all.

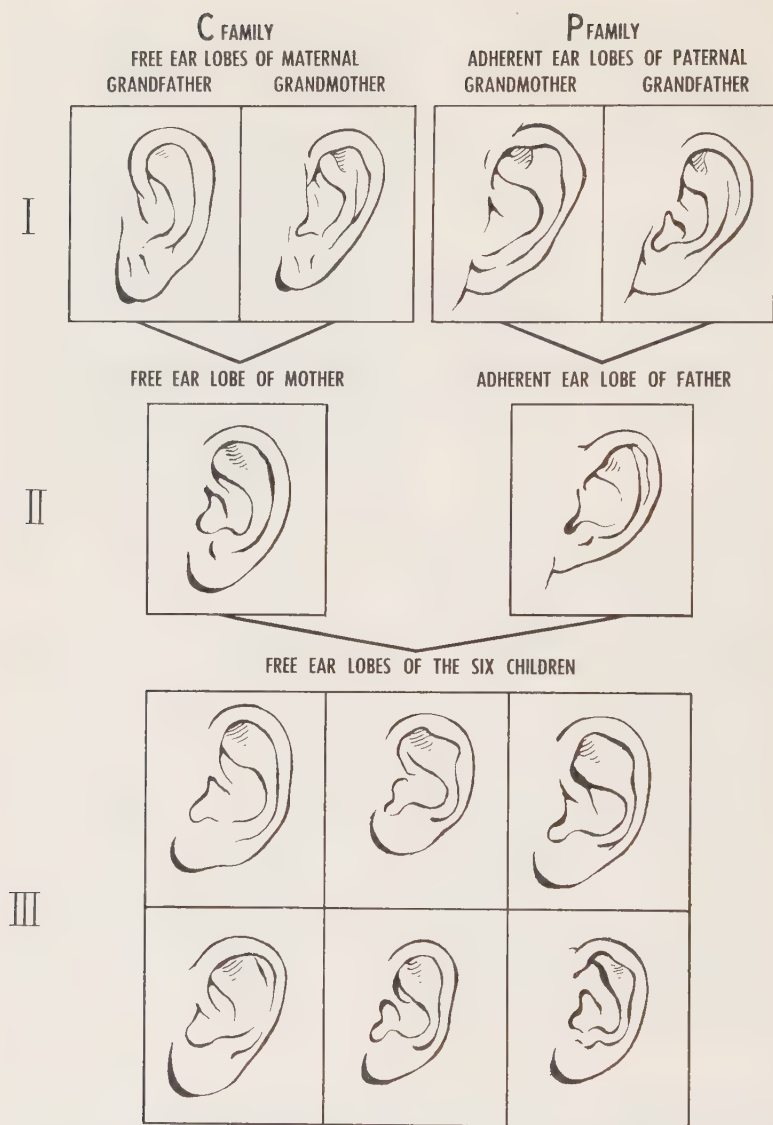


FIG. 35. A Family Pedigree of the Inheritance of Dominant Free and Recessive Adherent or Attached Ear Lobes. (From photographs after Powell and Whitney.)

Various other substances are similarly experienced or not. The ability to taste P.T.C. is due to a dominant gene; the inability to taste it is due to a recessive gene. There are remarkable differences among the ethnic groups of mankind in their ability to taste P.T.C., as shown in Table XV.

Ears

There are many different features of the ears which can be inherited independently, indicating that a number of genes are involved in determining the form of the ears. Size may vary considerably, whether the rims are rolled or flat, whether a tubercle is present in the upper margin of the rolled portion of the ear, whether a pit is present at the upper part of the front of the ear, and so on. Pits behave as irregular dominants. The lobe of the ear may hang free, and this form is determined by a dominant gene. The lobe that is attached to the head is inherited as a recessive. Large ears are dominant over small ears.

Deformed or misshapen ears are frequently associated with malformations of the organs of the genitourinary system. Frequently a single kidney is seriously affected, but anything from partial to complete failure of development may affect any one or more parts of the system. The condition appears to be inherited as a dominant with about 70 percent or more penetrance. Ear deformity can also occur without malformation of the genitourinary tract, but a malformed ear in the presence of enuresis, difficulty of micturition, abdominal colic, or other symptoms referable to the genitourinary tract indicates immediate investigation of the latter.

The Body

Stature

STATURE is a complex thing, and growth in height is dependent upon a large number of genes, for height is made up of growth in a vertical direction involving the segments of the lower extremities, the hips, the various vertebrae of the sacrum and back and their intervertebral discs, the neck, and the height of the skull. Some individuals may be extremely long-legged but have fairly short trunks; others may be short-legged and have long trunks; some have long necks and others short ones; still others have high heads while others have low ones. All these traits are controlled by genes and enter into the determination of stature. Interestingly enough there is some evidence that genes for tallness and shortness, as such, do in fact exist. Whether this be so or not, another interesting fact is that shortness appears to be rather more dominant than tallness, which, in effect, means that two short parents may have children of all heights, short, intermediate, and tall, because they may be carrying both "short" and "tall" genes, whereas tall parents will tend to have mostly tall children, for they carry paired genes for tallness in the recessive state and are unlikely to be carrying any "short" genes.

It should be fairly clear that if so many different segments of the body enter into the development of stature, this is a condition which must be markedly subject to the influences of the environment. And, indeed, this is precisely what we find—as we have already seen in Chapter 7. Socioeconomic environment, mainly as expressed through the nutritional factor, plays a highly significant role in influencing the development of stature. The

average individual raised in a poor socioeconomic environment is likely to be several inches shorter than his offspring who have been raised in a satisfactory socioeconomic environment. When the environment has been fairly equal for parents and their offspring, then it is found that there is a significant correlation between parental stature and the adult stature of their offspring. Short adults are likely to come from short parents, and tall adults are likely to have had tall parents. However, in both tall and short families, the offspring tend to be taller than the shorter parent. You can try this experimentally by asking, in any sizable group of people, all those to raise their hands who are taller than the shorter parent. You will find that the majority will raise their hands. If you then ask those who are taller than the taller parent to raise their hands, you will find the number still appreciable but somewhat reduced.

Body Form

There are a large variety of body forms from short and broad to tall and scrawny. Again, the mode of inheritance of body form is quite complex because of the number of parts and genes involved. In general, however, it is clear that body form is inherited in much the same way as stature and is subject to much the same environmental influences. For example, obesity is often due to an excessive intake of food, while leanness is often due to an inadequate intake of food. In addition, obesity and leanness are frequently the result of pathological disorders of a fairly large variety, mostly those affecting the endocrine glands. There is, however, no doubt that obesity and leanness, and most of the conditions in between, as well as body form are traits which may be conditioned by genes, regardless of the diet. There are whole families in which the body form is much the same in all or most of the members. The genetics of the subject is far from understood, but in some cases slenderness appears to be inherited as a recessive and obesity as a dominant, but in numerous other cases the mechanism of inheritance is not as simple as that.

There have been many different schemes by which the body has been classified, but not one of them has thus far proved satisfactory.

Being able to make a biologically sound, fairly reproducible estimate of the body type of an individual would enable us to

make studies on the relation of the body type to disease, temperament, immunity, and the like. Such studies have been made in recent years, but the results are quite inconclusive in spite of all claims to the contrary. Even the body types or somatotypes are quite arbitrarily standardized types. It is impossible to emphasize sufficiently the fact that all such studies are extremely difficult and must be viewed with the greatest caution.

If we are ever to make any progress in the understanding of the genetics of body form, it is the genetics of the different regions that will have to be studied rather than the body as a whole. The principal fault of most studies that have been attempted in the past is that they have attempted to treat the body as if it were inherited as a whole, when in fact it is inherited as a large variety of distinct units or components in interrelation. The interrelation of these distinct components forms the complex mosaic whole, the body. Because these components are the expression of the modified action of different groups of genes on different autosomes, their varieties and combinations in forming a morphologic whole are virtually unlimited. Hence, any attempt to describe body types on the basis of the gross description of the organism as a whole is foredoomed to failure. Certainly, the individual must be studied as a whole, but the description of that whole can satisfy scientific requirements only when the component parts which enter into its formation are analyzed and their interrelations properly understood. This is a task for a whole regiment of investigators rather than for the isolated student here and there.

Body Type and Temperament

Sheldon and others before and since have attempted to discover whether there is any relationship between body type and temperament. It is generally agreed that they have failed to do so.

The riddle of physique and temperament is one that bristles with unsolved problems. It is also complicated by the fact that body type changes with different ages. In adolescence many girls pass through a fat stage which they lose in the years prior to middle age and then, often, begin to reacquire. But some never do. The same thing often happens in boys. Bauer has shown that with age the chest tends to become more lateral in type, and there

is a tendency toward abdominal fat. The superficial soft parts of the body are well known to be highly subject to environmental influences. Nutrition, occupation, exercise, and numerous other environmental factors will make no difference to a man's blood groups or the shape of his nose, but such conditions will, to varying degrees, affect the size, proportions, and fatty development of the person.

The fact is that the more measurable traits that are included in any attempt to group men together, the more strongly emphasized does the essential individuality of the person become.

In man no genetic relationship has been described between body form or any part of the body and temperament. In rats evidence has been adduced which indicates that the Norway rat, which inherits a black coat color (nonagouti), tends to be tamer and less aggressive than the agouti or gray-haired segregates. In these rats the suggestion is that the effect of the genes upon behavior is pleiotropic, that is, it is the result of the action of many genes rather than being due to linkage with specific genes. Nothing comparable to such pleiotropic effects of genes associated with morphological traits is known in man, except in certain pathological and abnormal conditions.

Dwarfism

Extreme reduction in height and other dimensions of the body is usually caused by genetic factors. There are two main forms of dwarfism, the strong-man or *achondroplastic* form and the Tom Thumb or *ateliotic* form.

ACHONDROPLASTIC DWARFISM. Achondroplasia (Greek *chondros*, cartilage; *plasis*, a molding) refers to a failure in development of the cartilaginous portions of the bones, resulting in a premature union of the cartilage with the bone. In achondroplastic dwarfism the bones of the upper and lower extremities, and the base of the skull, are particularly affected. The arm bone (the humerus) and the thigh bone (the femur) are especially affected, so that the trunk and head seem disproportionately large in relation to the extremities. The arms give a curved appearance because they cannot be straightened at the elbow, and the legs are often bowed, so that the individual tends to waddle when he walks. Achondroplastic dwarfism seems always to be



FIG. 36. Three Types of Dwarfs with a Normal Man for Comparison.

	Age	Height
Cretin	30	2 ft. 11½ in.
Midget (Ateliotic dwarf)	20	3 " 3 "
Midget (Ateliotic dwarf)	28	3 " 4½ "
Achondroplastic dwarf	27	3 " 9 "
Achondroplastic dwarf	47	4 " 0 "
Normal man	—	5 " 6½ "

(From Rischbieth and Barrington.)

inherited as a dominant, and is believed to be due to a single dominant gene. Its occurrence in families in which there is no previous history of dwarfism indicates that achondroplastic dwarfism comes into being as a mutation in about 1 out of every 25,000 sex cells.

Achondroplastic dwarfs are of normal intelligence, and in medieval times were often employed as court jesters and sometimes attained positions of eminence. They also often served as models to painters. Achondroplastic dwarfs are perfectly fertile,

and produce an equal number of normal and dwarf children. The women have to be delivered by Caesarian section owing to the smallness of the contracted pelvis.

Among domestic animals the dachshund is an example of an achondroplastic dwarf.

ATELIOTIC DWARFS. Ateliosis (Greek *ateles*, incomplete) dwarfism is essentially characterized by a failure of the body to grow normally. The skeleton tends to remain in its infantile state. Cartilage in many parts of the skeleton fails to develop into bone, and some of the growing portions of the skeleton, the epiphyses, often fail to appear at all. These are the midgets. Three types have been distinguished: (1) a rare fetuslike midget never exceeding thirty-six inches in height, (2) true midgets, and (3) miniatures, who are essentially miniature adults who have never attained adult proportions, but have retained their infantile stature. Charles I of England's famous midget, Jeffery Hudson, was only eighteen inches tall when he was thirty years old. The famous Tom Thumb, a true midget, who was born in 1838, commenced his association with Barnum when he was four years old, at which time he was less than twenty-one inches in height. He grew, however, until in adult life he was somewhere in the vicinity of three feet tall. He married a dwarf, who bore him a child, which, alas, died in infancy.

Ateliotic dwarfism appears to be inherited in most cases as a simple recessive, due to a single recessive gene. Ateliotics seldom attain a height of forty-five inches, are sometimes achondroplastic, and frequently sterile. They are usually of normal intelligence, though sometimes of dull or defective mentality. Ateliotic dwarfism occurs rather less frequently than the achondroplastic variety.

Pygmies

Pygmies are members of populations in which the height of the individual does not exceed five feet. There are several ethnic groups of Pygmies: the African Pygmies or Negrillos of Equatorial Africa; the Asiatic Pygmies or Negritos, including the Andamanese of the Andaman Islands off the tip of southern India, the Semang of the central region of the Malay Peninsula and East Sumatra, and the Negritos of the Philippine Islands; and the Oceanic Pygmies or Negritos, the New Guinea Pygmies.

While it has been claimed that there is some evidence of achondroplasia in the Congo Pygmies this has never, in fact, been demonstrated, nor have there been adequate genetic studies of stature in any group of Pygmies. It is at present assumed that a mutant gene or genes occurred independently in these small isolated populations and rapidly established itself, resulting in a short-statured population. Precisely what aspects of the skeleton have been affected remains to be determined.

Cretinism

We have already discussed cretinism (see page 103) as a disease due to a deficiency in the hormonal secretion of thyroxin from the thyroid gland, and also as arising from environmental deficiencies of iodine in the soil. Thyroid deficiency in the mother during pregnancy is a frequent cause of cretinism in her offspring. However, it seems clear that there is a greater susceptibility to develop cretinism under iodine-deficient conditions in some individuals than in others, and this would suggest the possible presence of a recessive gene which renders its possessors more susceptible to cretinism than those who do not possess the gene. Early treatment with thyroxin greatly benefits the cretinous infant, though it seldom succeeds in restoring him to complete normality. The cretin usually remains very short in stature and severely retarded mentally.

Gigantism

Gigantism is usually due to an oversecretion of the growth hormones from the pituitary gland, and this is a pathological condition. Such a condition is rarely inherited, but cases are on record of extreme tallness affecting most members of a single family. Scheinfeld has described a case of a hereditary, non-pathological giant who was 7 feet 4 inches tall. His maternal grandfather attained 6 feet 7 inches, his father 6 feet 4 inches, his mother 6 feet, two brothers were 6 feet 10 inches and 6 feet 4 inches, and a sister was 6 feet 4 inches. Extreme tallness or gigantism when it is nonpathological would appear to be due to a rare recessive gene, which seems to have been carried by the parents of Jacob Nacken shown in figure 37.



FIG. 37. Dwarfs and Giants. The first four, three sisters and their brother, are midgets, aged respectively (from left to right) 38, 50, 34, and 48. Born in Dresden, Germany, of normal parents, they have four normal-sized sisters. The two men in the front row are achondroplastics, aged 42 and 31 respectively. The "giant" is 7 feet 4 inches, and 44 years of age. Tallness runs in his family. The "fat lady" weighs 540 pounds. Her condition appears to be glandular, for all the members of her immediate family are normal. (Courtesy of Amram Scheinfeld, from *The New You and Heredity*.)

The human species shows great variability in the distribution of the genes influencing stature. Some ethnic groups show much greater variability than others. For example, Europeans on the whole show greater variability in stature than Pygmies. The Japanese would appear to exhibit a greater frequency of small-stature genes than do Europeans, though there is good evidence that much of the shortness of many Japanese is due to environ-

mental factors. Greulich, for example, has recently compared the stature and weight of 898 American-born Japanese with a similar number of children in Japan and found the American-born Japanese to be strikingly taller and heavier than the children in Japan. Nevertheless, there can be no doubt of the reality of genes for shortness and tallness.

Nilotic Negroes exhibit a very high frequency of genes for tallness. So does the Hamitic people known as the Watusi of Central Africa who live in the vicinity of the northeast shore of Lake Tanganyika. Here both men and women are seldom under six feet tall while many are over seven feet tall. The genes for tallness are well distributed in this group and have enabled them to maintain an aristocratic dominance over the surrounding tribes for centuries. Their contempt for both the white man and other non-Watusi is unconcealed. Among their other accomplishments they happen to be spectacular high jumpers, exceeding their own height at each jump with ease. Among the Pygmies genes for shortness established themselves in an isolated population, and among the Nilotic Negroes and the Watusi the opposite genes for tallness established themselves in these isolates, whereas among the much mixed and mixing, nonisolated Europeans such genes have never had the opportunity to become fixed—there has been too much infusion of new genes all the time for such a thing to happen. Hence, when one plots the distribution of stature for a European population one finds that it assumes the normal or bell-shaped distribution curve. It is found that the majority of the population occupy an intermediate position at the top of the curve, being neither very tall nor very short, while the short and the tall occupy the lower parts of the curve.

The Head

The size and the shape of the head may be independently inherited. Broad heads (brachycephalic) appear to be dominant to the long heads (dolichocephalic). Mongoloids tend to be brachycephalic, and Negroids tend to be dolichocephalic. (A rough statement of the ethnic distribution of head shapes will be found in figure 26, page 209.) Every form of head shape is encountered among whites, although among some peoples, such as the populations inhabiting the Alpine regions of Europe and therefore

called the Alpine type, the head form shows a high frequency of brachycephaly. Scandinavians show a large frequency of long-headed forms. Evidently the genes for head form are variously distributed in frequency among the ethnic groups of mankind.

In individual families it is observed that broadheadedness tends to dominate longheadedness and that narrowness of the head tends to be recessive to longheadedness. However, as we have seen in an earlier chapter, head-shape genes are subject to the influence of the environment, the U.S.-born children of longheaded immigrant parents tend to have broader heads than their parents, while the U.S.-born children of broadheaded immigrant parents tend to have slightly narrower heads. The reason for this remains obscure.

People often ask whether the shape of the head can be permanently altered by artificial means. The answer is that it most certainly can. Children who lie on cradle boards often develop a flat back of the head. In fact, anthropologists who, not being aware of the fact that this occurred among Armenians in their homeland, brought into being a whole new "race," the so-called "Armenoid Race," which was characterized by a flat occiput. Armenians living in America discard the cradle board and cease to have flat occiputs! Many American, Middle American, and South American Indian tribes habitually practice artificial cranial deformation upon their infants, whose bones then change form in adaptation to the types of pressures to which they have been exposed. The shape of the head then remains permanently changed after the pressures have been removed.

The next question that is asked is, Are such artificially induced changes in the skull capable of being transmitted to the offspring of such persons? The answer is that they are not. Artificial cranial deformation has been practiced for thousands of years without in any way affecting the genes. It has been widespread in Africa from early times and was practiced among the ancient Egyptians without in any way affecting the heredity of these peoples. *Artificially acquired characters are simply not inherited, no matter what the trait may be that is in question.* For a trait to be inherited, there must be some genetic factor involved. If such a factor is not involved, the trait cannot be due to heredity.

THE FOREHEAD. The form and characters of the forehead tend to be inherited quite markedly. This involves such features as the height of the hairline, the presence of temporal hair, slants,

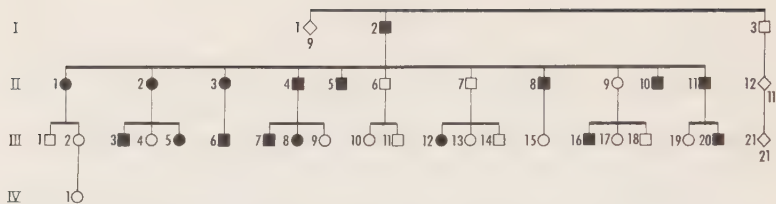


FIG. 38. A Pedigree of Extreme Susceptibility to Nasal Sinus Infection. (After Fraser Roberts, *An Introduction to Medical Genetics*.)

heights, widths, bulges, bosses (knobs), and supraorbital crests (crests above the eyes). These are quantitative traits, and it appears to be the general case that the larger quantitative expression of these forehead traits is due to dominant genes, the smaller expression to recessive genes.

The genes for frontal bosses, the paired bulges on the forehead, seem to be more frequently distributed among Negroes than among whites.

It remains to be said once more that none of these forehead features is in any way related to intelligence.

OCCIPITAL PROTUBERANCE. At the back of the head one can often feel a projecting area of bone. This is known as the *occipital protuberance*. It appears to be inherited as an incomplete dominant.

SINUSES. So far as I know no one has studied the inheritance of the sinuses of the head. Even now the function of these sinuses is not understood. They are, according to some theories, resonating chambers for the voice; according to others, they exist simply to lighten the weight of the head. Still others claim that they exist to provide ear, nose, and throat doctors with a living. I suspect that when the full story is in, it will be found that large sinuses are inherited as dominants of one kind or another, and that small ones are inherited as recessives.

The heredity of disease of the sinuses has, however, been studied in some striking cases. A case was reported by Grunberg, cited in Fraser Roberts, of a man who suffered from chronic inflammatory disease of the nasal sinuses. His ten brothers and sisters were completely free of the disease, as were their offspring and grandchildren, though among the latter there was one male

who suffered middle-ear disease (acute otitis media), and one who is said to have suffered from catarrh of the frontal sinuses. The brother originally exhibiting the disease, however, transmitted the susceptibility to ten males and six females in two generations. (See fig. 38.) Thus the susceptibility to acute inflammatory disease of the paranasal sinuses was inherited in one line as a dominant. In the other line of the healthy brother, the descendants had not inherited such a genetic susceptibility, and so were free of the disease in spite of the fact that they lived in similar environmental conditions in the same towns of western Germany.

Heredity and Blood

BLOOD is a complex tissue. Many constituents enter into its formation. Each of these constituents is genetically determined, and the interaction of these constituents gives rise to characteristics of the blood which are widely and variously distributed among the ethnic groups of mankind. The study of the distribution of these genetically determined characteristics of the blood is proving helpful in studying the relationships of ethnic groups to one another. But more important than that is the practical use to which we are able today to put our growing knowledge—and that knowledge literally increases daily—of the characteristics of the blood. One of the most familiar of the uses to which that knowledge is put is in blood transfusion—a use of knowledge which has already helped to save millions of lives. The characteristics of the blood are also helpful in assisting us to determine whether or not a particular individual can possibly be the parent of a particular child, in forensic medicine by identifying the characteristics of the blood from stains, and by typing the blood of potential parents prior to treatment which would otherwise have been overlooked and without which the life of the fetus would have been endangered.

The A-B-O Blood Groups

The A-B-O substances which determine the four principal blood groups were discovered by the Austro-American scientist Karl Landsteiner in 1900, and he distinguished them by the letters

TABLE XVI
THE AGGLUTINOGENS AND AGGLUTININS OF BLOOD GROUPS
A, B, AB, AND O

Blood Group	Agglutinogen (in red corpuscles)	Agglutinin (in serum)
AB	A and B	None
A	A—	anti-B
B	—B	anti-A
O	None	anti-A and anti-B

A, B, AB, and O. The four blood groups are determined by the fact that the red blood corpuscles (erythrocytes) contain two different antigens denoted by the letters A and B. An *antigen* is a substance which when injected into the blood of an animal results, after some time, in the appearance of antibodies in its blood serum. The antibodies are known as *agglutinins*, and the antigens which produce them are known as *agglutinogens*. The agglutinogens or agglutinative substances can be present either singly as in blood group A or blood group B, or together as in blood group AB, or be altogether absent as in blood group O.

The red corpuscles containing the agglutinogens float in the blood serum. In the presence of certain other serums the agglutinogens in blood groups A, B, and AB cause the red blood corpuscles to form clusters or clumps, that is, to agglutinate. The agglutination is produced by the two agglutinating substances, the agglutinins anti-A and anti-B, which are found in the blood serum of some persons. Once the blood corpuscles are agglutinated the agglutinins then proceed to destroy (hemolyze) them.

If a person is of the same blood group as another with whose blood serum some of his own blood is mixed, the blood corpuscles will generally disperse themselves evenly. This is due to the fact that members of the same blood group do not carry substances which would agglutinate their own blood. This is illustrated in Table XVI.

It should be perfectly understandable why the blood serum could not, and does not, normally carry substances that would cause its own red blood cells to agglutinate, for it would not do to have one's blood cells sticking to each other in circulating through the body. The Landsteiner Rule, therefore, is that if an agglutinogen is absent from the red blood corpuscles of a person, then the

TABLE XVII
DETERMINATION OF BLOOD GROUPS WITH TWO TEST SERA,
ANTI-A AND ANTI-B

	Known Serum Anti-A (Blood Group B)	Known Serum Anti-B (Blood Group A)	Blood Group
Agglutination of the un-	—	—	O
known blood corpuscles	+	—	A
	—	+	B
	+	+	AB
+ = clumping of red cells	— = no clumping		

corresponding agglutinin is present in the serum of that person. In blood transfusion it is important to avoid introducing blood containing agglutinogens that can react with agglutinins present in the serum of the recipient, for the introduced blood would then be destroyed or agglutinated, blocking the kidneys and even causing the death of the recipient. It will readily be seen that the blood groups can be determined by testing the unknown blood corpuscles with anti-A and anti-B sera, or by allowing the unknown serum to act on known corpuscles of A and B. The manner in which this may be done is shown in Tables XVII and XVIII. In Table XIX is shown the usual effect which follows upon adding a donor's blood to a receiver's serum.

In populations of European origin the commonest type of blood is O, a condition occurring in about 40 percent of the population. Since blood group O contains no agglutinogens, it was

TABLE XVIII
DETERMINATION OF BLOOD GROUPS OF SERA WITH KNOWN BLOOD
CORPUSCLES A AND B

	Known Corpuscles A	Known Corpuscles B	Blood Group
Agglutination by the un-	+	+	O
known serum	—	+	A
	+	—	B
	—	—	AB
+ = clumping of red cells	— = no clumping		

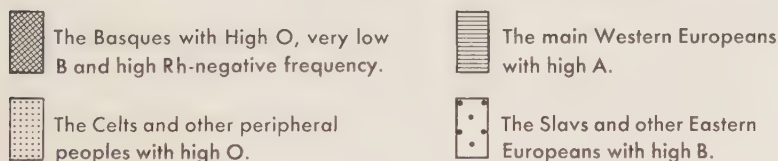
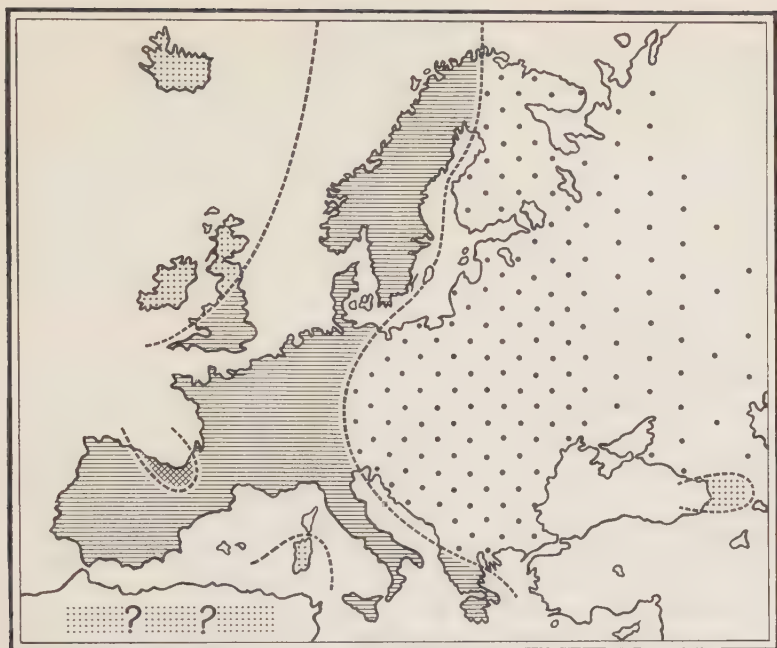


FIG. 39. Blood Groups in Europe. (After Chalmers, Ikin, and Mourant.)

formerly given to receivers irrespective of their blood groups; persons of blood group O were therefore called “universal donors.” The ab agglutinins of blood group O are generally rendered harmless by dilution or other mechanisms when group O blood is transfused to persons of other blood groups. It happens, however, that some persons of blood group O possess agglutinins of exceptionally high clumping power; the use of their blood is therefore dangerous since, even when diluted, it may destroy the red blood corpuscles of persons of other blood groups. For this reason donors of the same blood group are, whenever possible, used.

Since persons belonging to the least common blood group, AB, about 5 percent of the population, possess no agglutinins they were at one time considered to be capable of receiving the blood of any other group, and were therefore called "universal recipients." But the red blood corpuscles of such "universal recipients" were occasionally clumped by the introduced donor's agglutinins. For these reasons the use of so-called "universal donors" and "universal recipients" is restricted today to special cases. Blood group A occurs in about 40 percent of persons of European stock and blood group B in from 10 to 15 percent.

Three genes are responsible for the four blood groups, these genes are designated by the italicized letters *A*, *B*, and *O*. There is only one gene on each chromosome for the agglutinable properties of the red corpuscles. At fertilization the double number of chromosomes is produced and two genes for the blood groups now go into the making of each individual. There are now six possible pairs of genes that the individual can inherit, as shown in Table XX, *AA*, *AO*, *BB*, *BO*, *AB*, and *OO*. The blood group to which a person belongs depends upon which pair out of these six possible pairs of genes he has inherited from his parents, only one pair of which each parent himself can have possessed. The genetic constitution of human beings with regard to the blood groups is therefore determined in the manner shown in Table XXI.

Genes *A* and *B* are of equal expressive value and therefore the substances which they determine occur together as recognizable

TABLE XIX
THE USUAL EFFECT OF ADDING A DONOR'S BLOOD TO A RECEIVER'S SERUM

Agglutinins in receiver's serum	Agglutinogens of Donor's Corpuscles			
	(Group A) A	(Group B) B	(Group AB) AB	(Group O) None
(Group A) anti-B	Compatible	Agglutinated	Agglutinated	Compatible
(Group B) anti-A	Agglutinated	Compatible	Agglutinated	Compatible
(Group AB) none	Compatible	Compatible	Compatible	Compatible
(Group O) anti-A, anti-B	Agglutinated	Agglutinated	Agglutinated	Compatible

TABLE XX
THE GENE COMBINATIONS OR GENOTYPES YIELDING THE PHENOTYPES
OR BLOOD GROUPS

Genotype	Phenotype
<i>AA</i> or <i>AO</i>	A
<i>BB</i> or <i>BO</i>	B
<i>AB</i>	AB
<i>OO</i>	O

agglutinogens. Gene *O* is masked by or is recessive to *A* and *B*, so that *O* is not expressed in the presence of the alleles *A* or *B*. Thus, for a person to belong to group *O* both of the parents must have carried the gene, either in a homozygous condition, where both genes were alike, or in a heterozygous condition, where one gene in each parent was *O* and the other either *A* or *B*. In the former event, all the children would belong to blood group *O*, as for example is the case among such South American Indian tribes as the Chulupie of Argentina, the Guarani of Paraguay, and the Onas, Yámanas, and Alakalufs of Tierra del Fuego. Where *O* is carried by both parents in the heterozygous state, the offspring could belong to any one of the four blood groups. The mode of transmission of the genes in the latter case is illustrated in figure 40. From these facts it will be seen that the blood-group genes yield six genotypes and four phenotypes as shown in Table XX.

The account given of the blood groups in the preceding paragraphs is accurate as far as it goes, but in fact blood group *A* is known to us in two forms, each with its own gene, designated *A*₁ and *A*₂ with *A*₁ apparently dominant over *A*₂, so that there are four rather than three genes involved in the genetics of the blood groups, giving rise to ten genotypes. The recognition of these subgroups are of the greatest value, for by their means our understanding of the problems which they can help to solve is rendered so much more refined. We cannot, however, develop the significance of the subgroups further here.

The Secreting Factor

The A-B-O blood-group factors may also be determined from the natural secretions—the saliva, gastric juices, mucous secre-

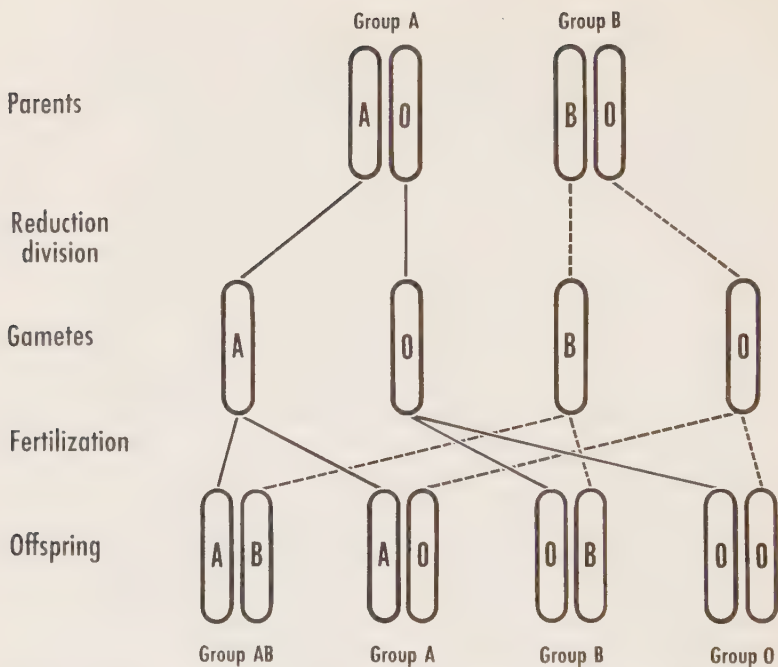


FIG. 40. Chromosome diagram showing the transmission of the genes in the mating of two persons, one of blood group A and the other of blood group B, each being heterozygous for blood group O.

tions, and urine—of some persons. Such persons are termed “secreitors.” Persons whose secretions are nearly free of these factors (in water-soluble form) are termed “nonsecreitors.” The heredity of the secreting factor is extremely simple, two genes being involved, one of which is dominant, *S*, and the other is recessive, *s*, thus giving rise to three genotypes, as follows:

<i>Genotype</i>	<i>Phenotype</i>
<i>SS</i>	Secretor
<i>Ss</i>	Secretor
<i>ss</i>	Nonsecretor

Altogether apart from its value in studies of heredity, the possibility of determining the blood factors from such secretions has enabled experts in rare cases to bring more than one criminal to

TABLE XXI
GENETIC CONSTITUTION WITH REGARD TO BLOOD GROUPS

Sperm containing chromosome carrying gene*	Ovum containing chromosome carrying gene†	Genotype	Blood Group (Phenotype)
<i>A</i>	<i>A</i>	<i>AA</i>	A
<i>A</i>	<i>O</i>	<i>AO</i>	
<i>O</i>	<i>A</i>	<i>OA</i>	
<i>B</i>	<i>B</i>	<i>BB</i>	B
<i>B</i>	<i>O</i>	<i>BO</i>	
<i>O</i>	<i>B</i>	<i>OB</i>	
<i>A</i>	<i>B</i>	<i>AB</i>	AB
<i>B</i>	<i>A</i>	<i>BA</i>	
<i>O</i>	<i>O</i>	<i>OO</i>	O

* These genes could be carried by chromosomes in the ovum.

† These genes could be carried by chromosomes in the sperm.

justice from the evidence of his hereditary traits as carelessly left behind him on a discarded cigarette butt or on an envelope!

Blood Types M, N, and MN

Some thirty years after the discovery of the A-B-O blood groups, Dr. Landsteiner and his colleague, Dr. Phillip Levine, described another system of blood types which depended upon two antigens, called M and N and determined by a single pair of genes. When a chromosome in which gene *M* is located pairs with a chromosome containing gene *M* the resulting blood type is M. When a chromosome containing gene *M* pairs with a chromosome with the *N* gene the resulting blood type is MN. When pairing is between chromosomes containing *N* genes at each of their corresponding loci, the blood type is N. This is clearly brought out in Table XXII.

The M-N system is quite independent genetically of the A-B-O system, and no one thus far examined has lacked these M-N antigens, which are inherited as characters without dominance. Hence, it is a simple matter to work out the proportion of M-N

types in children born to parents whose M-N types are known. Try it before looking at anything but the parents' blood types in Table XXIII.

In man the M and N agglutinogens or antigens seldom have any natural agglutinins or antibodies; hence, it is hardly ever necessary to take them into consideration when making transfusions. The test sera are obtained from rabbits which have been injected with human blood of types M and N, and which have developed antibodies against these antigens.

An important discovery by Sanger and Race made in 1947 of a new antibody intimately associated genetically with the M-N system has served greatly to enlarge the usefulness of this system for genetic analysis. This new antibody was found to agglutinate 72 percent of type M samples of red corpuscles, 60 percent of MN samples of red corpuscles, and 33 percent of type N samples. The agglutinin or antigen thus agglutinated has been assigned the letter *S* for the dominant gene and small *s* for the recessive gene. The system, therefore, becomes the M-N-S-s blood-type system controlled, according to Sanger and Race, by two pairs of closely linked genes, with six phenotypes, or blood groups, and ten genotypes, as shown in Table XXIV. Wiener, however, considers that multiple rather than linked genes are involved—the genes L^A , L , I^A , and I —with nine phenotypes and ten genotypes, as shown in Table XXV.

TABLE XXII
THE HEREDITY OF BLOOD TYPES M, N, AND MN

Sperm containing chromosome carrying gene*	Ovum containing chromosome carrying gene†	Genotype	Blood Type
<i>M</i>	<i>M</i>	<i>MM</i>	M
<i>M</i>	<i>N</i>	<i>MN</i>	MN
<i>N</i>	<i>M</i>	<i>MN</i>	
<i>N</i>	<i>N</i>	<i>NN</i>	N

* These genes could be carried by chromosomes in the ovum.
† These genes could be carried by chromosomes in the sperm.

TABLE XXIII
THE INHERITANCE OF THE M-N BLOOD-GROUP SYSTEM

Parents	Children		
$MN \times MN$	$\frac{1}{4}MM$	$\frac{1}{2}MN$	$\frac{1}{4}NN$
$MN \times MM$	$\frac{1}{2}MM$	$\frac{1}{2}MN$	
$MN \times NN$		$\frac{1}{2}MN$	$\frac{1}{2}NN$
$MM \times MM$	All MM		
$MM \times NN$		All MN	
$NN \times NN$			All NN

The Rh Blood Types

The significance of the Rh factor has to some extent already been dealt with on pages 88-91. The reader is advised to reread those pages at this point and to return to this page for a more detailed discussion of the genetics of the Rh types.

Like the M-N-S-s series the Rh antigens do not normally have antibodies associated with them in the blood serum, thus differing from the A and B antigens which do. But the Rh antigens differ from the M-N-S-s series in that when they are introduced into the blood of an individual lacking in them, the Rh antigens will often induce the formation of antibodies. On a first transfusion no untoward results will occur if the donor and the recipient are alike on the A-B-O system. But on a second transfusion the antibodies which may be produced by the first transfusion will tend to agglutinate the red blood corpuscles, which may result in death.

TABLE XXIV
THE M-N-S-S BLOOD-GROUP SYSTEM

Phenotypes	Genotypes
MS	$MSMS$ or $MSMs$
Ms	$MsMs$
MNS	$MSNS$, $MSNs$, or $MsNS$
MsNs	$MsNs$
NS	$NSNS$ or $NSNs$
Ns	$NsNs$

TABLE XXV
NOMENCLATURE OF THE M-N-S-s TYPES

Three M-N Phenotypes			Six M-N-S Phenotypes		Nine M-N-S-s Phenotypes		
Designation	Reaction with serum		Designation	Reaction with serum	Designation	Reaction with serum	Corresponding Genotypes
	Anti-M	Anti-N					
M	+	—	MS	+	MSS	—	$L^s L^s$
					MSs	+	$L^s L$
					Mss	+	LL
N	—	+	NS	+	NSS	—	$I^s I^s$
					NSs	+	$I^s I$
					Nss	+	II
MN	+	+	MNS	+	MNSS	—	$L^s I^s$
					MNSs	+	$L^s I$ and LI^s
					MNss	+	LI

Adapted from Wiener, A. S., and Wexler, I. B., *Heredity of the Blood Groups*.

This mechanism has already been described in the case of maternal-fetal incompatibility.

That some individuals are born with an Rh factor (Rh-positive) and others not (Rh-negative) is entirely due to their heredity. It is now known that there are three principal Rh factors in the blood, the original **Rh₀**, which is by far the most powerful and clinically the most important, and the **rh'** and **rh''**. Type rh (triple Rh-negative) blood lacks all three factors, so that it can therefore be safely used in cases of intragroup incompatibility due to Rh factors.

The three elementary factors or antigens, **Rh₀**, **rh'**, and **rh''**, have three theoretically possible contrasting factors designated **Hr₀**, **hr'**, and **hr''**, of which, however, only **hr'** and **hr''** have been found to exist. With rare exceptions, every blood contains at least one factor of each of the pairs of genes *rh'-rh'*, and *rh''-rh''*. The Hr factors are less antigenic (that is, less capable of stimulating the formation of specific reacting substances) than the Rh factors. The three elementary Rh factors, it is now known (together with the Hr factors **hr'** and **hr''**), determine eight agglutinogens. Since the chemical constitution of the agglutinogens is unknown, they cannot be determined by chemical analysis, but their presence can be identified by their reactions with specific immune sera.

TABLE XXVI
RH GENOTYPES AND PHENOTYPES

Union of		Genotype	Rh Phenotype
Sperm containing chromosome carrying gene*	Ovum containing chromosome carrying gene†		
r	r	rr	rh
r	r'	$r'r$	rh'
r'	r'	$r'r'$	
r	r''	$r''r$	rh''
r''	r''	$r''r''$	
r'	r''	$r'r''$	rh'rh'' (rh _y)
r	R^0	R^0r	Rh ₀
R^0	R^0	R^0R^0	
r	R^1	R^1r	Rh ₁
R^0	r^1	R^0r^1	
R^0	R^1	R^1R^0	
r^1	R^1	R^1r^1	
R^1	R^1	R^1R^1	
r	R^2	R^2r	Rh ₂
R^0	r''	R^0r''	
R^0	R^2	R^2R^0	
r''	R^2	R^2r''	
R^2	R^2	R^2R^2	
r'	R^2	R^2r'	Rh ₁ Rh ₂ (Rh _z)
r''	R^1	R^1r''	
R^1	R^2	R^2R^1	

* These genes could be carried by the chromosomes in the ovum.

† These genes could be carried by the chromosomes in the sperm.

Adapted from Montagu, A., *An Introduction to Physical Anthropology*.

Each of the eight agglutinogens thus recognized is determined by a particular dominant gene. This dominance means that if an agglutinogen is present in an individual, then that particular agglu-

tinogen must be present in at least one of the parents. A single agglutinin may have anything from one to three factors and therefore may react with as many sera. One genotype, $R^z r$, will react with all six antisera, which contain the antibodies, being divided into two subclasses, Rh and Hr. The eight allelic genes for the agglutinogens are written R^o , R^1 , R^2 , R^z , r , r' , r'' , r^v . Since every person has but one pair of Rh genes, a single one derived from the maternal pair and one derived from the paternal pair, there are thirty-six possible ways in which the eight genes can be combined. Using the six most frequently occurring genes (R^z and r^v being very rare), there are twenty-one possible ways in which these six genes can be combined. In other words, twenty-one genotypes are possible which express themselves in eight Rh blood types or phenotypes. The Rh type of the individual is therefore the expression of his genotype determined by a single pair of genes, each allele or member of the pair being derived from the opposite parent. The twenty-one possible genotypes produced by this series of six pairs of genes and the eight blood types to which they give rise are shown in Table XXVI. The frequency of the Rh genes and their corresponding agglutinogens as found among whites in the city of New York by Wiener, together with the positive reactions (+) and negative reactions (—) to the Rh and Hr sera, is shown in Table XXVII. It is a rule that in the case of homozygous individuals where there is a positive reaction to the serum of one set, there is a failure to react with the serum of the other set. On the other hand, in the case of a heterozygous individual his agglutinogens will react with sera in both sets.

The distribution of the Rh genes among the ethnic groups of mankind is extremely interesting and most helpful in indicating possible origins and relationships. In Table XXVIII are shown the percentage of individuals (rather than the percentage of genes) in the various ethnic groups in whom the various blood types occur. From this table it will be observed that the Basques have an extremely high frequency of Rh-negative, 28.8 percent. It is quite possible that the Rh-negative gene originated in Europe from a population of whom the Basques are the present-day representatives. However, there are small isolated populations in Switzerland that are characterized by even higher Rh-negative frequencies than the Basques. Such, for example, are the Western Walsers

TABLE XXVII
THE RH SERIES OF ALLELIC GENES *

Genes	Gene frequencies among New York City Caucasoids (percent)	Corresponding agglutinogens	Reactions with Rh serums †			Reactions with Hr serums†		
			Anti-Rh ₀	Anti-rh'	Anti-rh''	Anti-Hr ₀ (hypothetic)	Anti-hr'	Anti-hr''
r	38.0	rh	—	—	—	+	+	+
r'	1.4	rh'	—	+	—	+	—	+
r''	0.5	rh''	—	—	+	+	+	—
r ₇	0.01	rh ₇	—	+	+	+	—	—
R ⁰	3.2	Rh ₀	+	—	—	—	+	+
R ¹	40.4	Rh ₁	+	+	—	—	—	+
R ²	16.4	Rh ₂	+	—	+	—	+	—
R ³	0.1	Rh ₃	+	+	+	—	—	—

* After Wiener.

† A single pair of genes is responsible for the individual's phenotype, e. g., genotype R¹R² yielding Rh, Rh₂. By analysis of the phenotype in homozygous individuals the effect of a single gene may be inferred.

who in one village (Tenna) show a frequency of 39.5 percent Rh-negative. It will be seen that Mongoloids, Polynesians, Melanesians, and Australoids tend to be entirely Rh-positive, and that there are interesting differences between other groups. In figure 38 something of the pattern of distribution of the main blood groups in Europe is shown.

Other Blood Types

It is only possible to mention here some other blood types which have been discovered in recent years and which are under genetic control. These are:

The P antigen due to a dominant gene, *P*, with its recessive allele, *p*.

The *Kell type* due to an incompletely dominant gene, *K*, with the recessive gene designated *k*.

The *Lewis type* due to a recessive gene. The gene for this antigen behaves as a recessive in the adult, but exerts an effect in the infant only in the heterozygous state.

TABLE XXVIII
DISTRIBUTION OF THE RH BLOOD TYPES
(As Determined by the Use of 3 Sera: Anti-Rh₀, Anti-rh', Anti-rh'')

Population	Investigators	Number of Subjects Tested	(Rh +)	PERCENTAGES OF RH TYPES							
				Rh ₀	Rh ₁	Rh ₂	Rh ₁ Rh ₂	Rh ₀	rh'	rh''	rh'rh''
Papuan Admiralty Islanders	Simmons <i>et al.</i> (1946) Simmons & Graydon (1947)	100	100.0	0	93.0	0	7.0	0	0	0	0
Fijians	Simmons & Graydon (1947)	112	100.0	0	92.9	0.9	6.2	0	0	0	0
Filipinos New Caledonians	Simmons & Graydon (1945)	110 100	100.0 100.0	0 0	89.1 87.0	1.8 2.0	9.1 11.0	0 0	0 0	0 0	0 0
Loyalty Islanders Indonesians	Simmons & Avias (1949) Simmons & Avias (Incomplete)	243 103	100.0 100.0	0 0	77.4 77.7	2.1 2.9	20.5 19.4	0 0	0 0	0 0	0 0
Australian Aborigines	Simmons & Graydon (1947)	200	100.0	0	74.0	2.5	22.5	0.5	0	0	0.5
American Indians	Simmons <i>et al.</i> (1948)	234	100.0	0	58.2	8.5	30.4	1.3	1.7	0	0
(Mexico) American Indians	Wiener <i>et al.</i> (1945)	95	100.0	0	48.1	9.5	41.2	1.1	0	0	0
(Oklahoma) Maoris	Wiener <i>et al.</i> (1946)	105	100.0	0	40.0	17.1	39.1	2.9	0.9	0	0
Japanese	Simmons <i>et al.</i> (Incomplete)	32	100.0	0	25.0	31.0	41.0	3.0	0	0	0
Japanese	Miller and Taguchi	180	99.4	0.6	51.7	8.3	39.4	0	0	0	0
Chinese	Waller and Levine	150	98.7	1.3	37.4	13.3	47.3	0	0	0	0.7
Asiatic Indians	Wiener <i>et al.</i> (1944)	132	98.5	1.5	60.6	3.0	34.1	0.9	0	0	0
(Moslems) American Negroes	Wiener <i>et al.</i> (1945)	156	92.9	7.1	70.5	5.1	12.8	1.9	2.6	0	0
American Negroes	Wiener <i>et al.</i> (1944)	223	91.9	8.1	20.2	22.4	5.4	41.2	2.7	0	0
Puerto Ricans	Levine <i>et al.</i> (1945)	135	92.6	7.4	23.7	16.3	4.4	45.9	1.5	0.7	0
White, Americans	Torregosa <i>et al.</i> (1945)	179	89.9	10.1	39.1	19.6	14.0	15.1	1.7	0.5	0
White, Americans	Wiener <i>et al.</i> (1946)	766	87.5	12.5	54.7	14.9	14.0	2.2	0.9	0.5	0
White, English	Unger <i>et al.</i> (1946)	7,317	85.3	14.7	53.5	15.0	12.9	2.2	1.1	0.6	0.01
White, English	Fisher & Race (1946)	927	85.2	14.8	54.9	12.2	13.7	2.5	0.7	1.3	0
White, English	Murray (1946)	1,038	84.7	15.3	54.8	14.7	11.6	2.3	0.6	0.7	0
White, Australians	Simmons <i>et al.</i> (1945)	350	85.1	14.9	54.0	12.6	16.6	0.6	0.9	0.6	0
White, Hollanders	Graydon <i>et al.</i> (1946)	200	84.6	15.4	51.5	12.3	17.7	1.5	1.5	0	0
White, French	Simmons <i>et al.</i> (1946)	501	83.0	17.0	51.7	13.6	13.0	3.6	0.4	0.8	0
Basques	Bessis (1946)	383	69.5	28.8	55.1	7.8	6.0	0.6	1.8	0	0
Basques	Chalmers <i>et al.</i> (1949)	250	64.4	35.6	—	—	—	—	—	—	—
Swiss (Davos-Sertig)	Eicheverry (1947)	34	67.6	32.4	—	—	—	—	—	—	—
Swiss (Wiesen)	Moor-Jankowski & Huser (1958)	66	65.5	34.5	—	—	—	—	—	—	—
Swiss (Tenna)	Moor-Jankowski & Huser (1958)	38	60.5	39.5	—	—	—	—	—	—	—

Adapted from Montagu, A., *An Introduction to Physical Anthropology*.

The *Lutheran type* is due to a dominant gene, *Lu*.

The *Duffy type* is due to a dominant gene, *Fy^a*.

The *Kidd type* is probably due to a dominant gene, *Jk*.

In addition to all these "public" types of blood that are widely distributed through populations, there are also some "private" types of blood which occur occasionally in particular families. Some nine of the latter have been described, but with further research it may be found that these "private" antigens are not as limited in their distribution as would at the present time appear. However this may be, it is obvious that a great deal more research needs to be done in this fascinating and important field of genetic serology.

One of the great advantages for the study of heredity of the blood groups is that a particular gene is responsible for a particular antigen in the red blood corpuscles, whereas in such traits as skin color, hair form, and the like, several genes are involved. The mechanism of inheritance of a trait in which several genes are involved is much more difficult to unravel than that in which a single gene is at work. In the latter case we are often helped in solving the problems of the workings of heredity because the variables involved are few and comparatively clear-cut. A few examples will suffice to show how our knowledge of the blood groups helps us solve not only practical problems but also those of great theoretical interest.

When two things always occur in the presence of each other but are seldom found when the other is not also found with it, we have a right to suspect that they are connected. When this happens in the case of one trait in connection with a gene for some other trait, we may suspect that there is some sort of significant relationship between the genes for these traits. Such a relationship may be one of more or less close linkage or the expression of the multiple effects of a single gene.

Recently Drs. Renwick and Lawler in England have brought forward evidence indicating that the abnormality known as the nail-patella syndrome was linked with certain blood-group genes. Individuals affected by this disorder have poorly formed fingernails, particularly of the thumb and index finger, absent or small kneecaps (patellae), and sometimes dislocated elbows and horny growths on the pelvic bones. The condition is due to a single dominant gene. Drs. Renwick and Lawler studied an affected man of blood group A₁B who had conveniently married a perfectly normal woman of blood group O. They had had sixteen

children of whom eleven were living at the time of the investigation. Four of the children had inherited the father's B gene, and it was just these four, two girls and two boys, who showed the nail-patella syndrome. The other seven children had inherited their father's A_1 gene and were completely free of the disorder. It would appear, then, that the nail-patella gene is on the same chromosome as the B gene. Further study revealed that not all families showed such a close linkage, for in about 10 percent of cases there had been crossing over, which had separated the two genes. In some cases the nail-patella gene was found to be linked with the O and A_1 genes.

Linkages such as these not only enable us to make detailed maps of the distribution of particular genes on the chromosomes and to identify the relation to other genes on the same chromosomes, but they also enable us to predict the frequency with which such conditions will come into being in any family whose blood groups are known. In this way it is possible to prepare parents for problems, which being anticipated may be dealt with both practically and psychologically in a more satisfactory manner.

Blood Groups and Disease

In recent years a number of independent investigators have found evidence which indicates that persons belonging to different blood groups may differ substantially in their susceptibility to certain diseases of adult life. In 1951, Struthers in England drew attention to a significant relationship between the frequency of blood group A and bronchial pneumonia. In 1953 Aird, Bentall, and Roberts, comparing the blood groups of 3,632 individuals suffering from cancer of the stomach with a control series free of the disease, concluded that "the frequency of blood group A is greater and the frequency of blood group O less in patients suffering from cancer of the stomach than in the general population of the locality in which they live." For example, in Newcastle they found that blood group A occurred in 43.6 percent of the individuals with cancer, but in only 37.4 percent of the controls who were free of the disease. In other places the figures were as follows: Leeds, 47.9 cancer, 40.3 control; Manchester, 44.5 cancer, 38.4 control; Liverpool, 44.7 cancer, 39.6 control; Birmingham, 57.0 cancer, 44.4 control; London, 46.0, cancer 42.2 control; and Scotland, 36.4 cancer, 32.5 control.

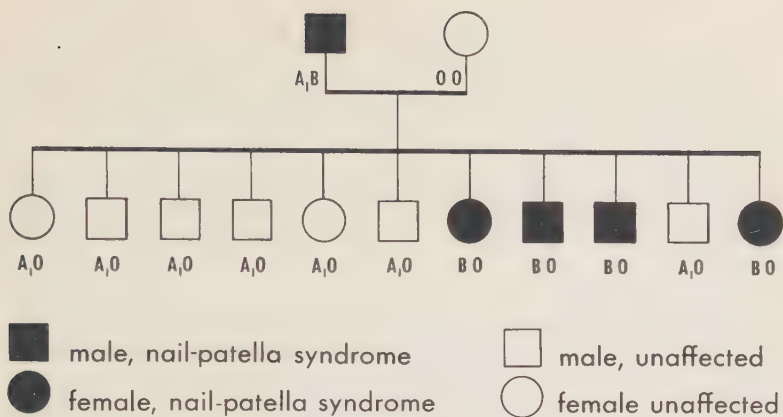


FIG. 41. Pedigree Showing Linkage Between A-B-O Blood Groups and the Nail-Patella Syndrome.

The consistency with which blood group A is in every case significantly more frequent in the cancer victims than in those free of the disease is impressive. However, Speiser in Vienna and Wallace in Glasgow were unable to find any association between cancer and the A-B-O groups in large series of individuals examined by them. Nor have other investigators been able to find such an association. On the other hand, Haddock and McConnell found a significantly high frequency of blood group A in cases in which the cancer arose in the body of the stomach as compared with growths affecting the pylorus or the antrum of the stomach. Clearly, further careful research is required.

In 1954 Aird and his associates claimed that in a series of 3,011 cases from three localities in England the frequency of blood group O was significantly higher in patients with peptic ulcers (that is, ulcers affecting either the stomach or duodenum) than in the control series. This relationship has since been confirmed by more than half a dozen different investigators. While there remains some question as to the significance of the association between blood group O and stomach ulcers, the association between duodenal ulcers and blood group O seems to be definitely proven.

There is some evidence that pernicious anemia occurs more frequently in persons of groups A than in those of group O.

In Scotland in 1956 McConnell, Pyke, and Roberts found that in men diabetes mellitus was significantly associated with a high frequency of blood group A, but in the case of women sufferers there was no significant correlation with any blood group.

Claims for the association of certain blood groups with other diseases have not thus far been substantiated. Such possible associations are at the present time being investigated by a number of different investigators, and we shall have to await their findings with patience.

Meanwhile, it cannot be too strongly emphasized that the association between blood groups and disease by no means implies a causal relationship. There is no evidence that it is blood of a certain type which produces the susceptibility to disease. It seems rather more likely that if an association exists between blood groups and disease—and this is denied by some authorities like Wiener and Wexler, and Manuila—the blood groups may simply represent indicators of some other factor or factors with which they are associated and which are more directly related to the susceptibility to certain types of disease—but what these other factors may be can at present be a matter for conjecture only. A clue to the possible factors involved has been suggested by Dr. A. J. Cain of Oxford. Dr. Cain points out that secretors pour a considerable amount of the antigens from their salivary glands into the commencement of the digestive system, the mouth. This at once suggests that something is being taken into the body, as part of the food, which it is advantageous to neutralize as quickly as possible. In 1948 Renkonen and in 1949 Boyd and Reguera independently discovered that the seeds of many leguminous plants widely used for food, such as the lima bean, contain large quantities of blood-group agglutinins which can be neutralized by secretors. This suggests that these antibodies may have a deleterious effect on some parts of the absorptive epithelial lining of the digestive tract. Cain points out that while such action may be of little importance to civilized man, it might have been of considerable significance under the far more strenuous conditions under which man lived in the prehistoric period. The finding of Clarke and others in 1956 that secretors had a considerably lower incidence of duodenal ulcer as compared with nonsecretors of the A-B-O blood-group antigens strongly suggests the actual nature of the deleterious action. Thus, secretors would have a definite selective advantage in this respect.

What Do We Do About Heredity?

Questions

ACHIEVING some understanding of the meaning of heredity, of the manner in which heredity works, is a necessary step in the understanding of some of the fundamental problems that concern man in society. How many children should we have? Should we have any children at all? Should incorrigible criminals or persons affected with certain disorders be sterilized? Is it not true that as a consequence of our immense progress in medicine and sanitation we have enabled untold numbers of unfit individuals to survive who not only would be far better off dead, but who constitute an increasing burden to society and at the same time serve to debase the quality of man's germ plasm? Is not the national intelligence of the country being lowered by the unregulated breeding of the unfit and the mediocre while the fit and the highly intelligent scarcely reproduce themselves? Should not an intelligent society regulate the breeding habits of its members, so that the best are encouraged to reproduce most and the least fit discouraged from breeding altogether? Isn't there some connection between color and culture? It isn't really possible, is it, to make silk purses out of sows' ears? What about birth control? These and a thousand like questions are the questions frequently asked by men and women in our society.

Ethics and Science

Our understanding of the facts of human heredity enables us to return sound answers to many of those questions, and in those

instances in which knowledge is lacking we can frankly recognize our ignorance and take the necessary precautions against uninformed judgments or rash prescriptions for action. In any event, it should be quite clear that what we do about applying our knowledge of human heredity to the solution of human problems, while it may be most strongly supported by a knowledge of the scientific facts, is in the first place a matter of ethics, and only secondarily a matter of science.

Ethics is concerned with standards of conduct and moral judgment, with the distinction between right and wrong, with leading, in short, the good life. What is right and wrong for human beings? What is the good life? Countless men have delivered themselves on these matters, and libraries of books have been written in the attempt to answer those questions. Without in any way desiring to slight the answers that have been traditionally given to these questions I will here offer an answer provided by a group of scientists in 1939. During the course of a discussion the question arose whether it might not be possible to discover a naturally operative principle which governs human conduct. The discussants, Professors Edwin Grant Conklin, C. Judson Herrick, Olaf Larsell, and Chauncey Leake, eventually arrived at the following general principle: "The probability of survival of individual, or groups of, living things increases with the degree with which they harmoniously adjust themselves to each other and their environment."

I have independently offered a principle which is in essential agreement with this. It is that goodness in human conduct consists in behavior which confers survival benefits in a creatively enlarging manner upon others. In reality I think the latter principle complements the first. "Harmonious adjustment" and "in a creatively enlarging manner" are the key ideas in these principles, once we take the desirability of survival for granted. What is meant by "harmonious adjustment" and benefits conferred upon others "in a creatively enlarging manner"?

We mean, quite simply, that human beings stand the best chance of realizing their potentialities when they live co-operatively together, using their natural and social inheritance wisely; that they do not become the spendthrifts of that inheritance, but that they utilize the gifts that both nature and society have brought to them to maximize the happiness of every member of

society. Hence, if overcrowding causes misery, disharmony, and renders the harmonic development of the person difficult, then *ipso facto* it must be considered an evil, whether the overcrowding be familial, communal, national, or on a world basis. Therefore even if all individuals were constitutionally healthy, overcrowding or overpopulation must be considered an evil because it renders the harmonious adjustment of the individual to his environment difficult. Therefore, we ought to regulate our numbers, on a familial, community, national, and international basis in such a manner that we would avoid the evils of overcrowding or overpopulation, and render possible the optimal development of the individual. As individuals and as a society we ought to think seriously about family limitation for those whose families ought to be limited and of ways of encouraging married couples to have children who ought to have many children. And we ought to think seriously also about the problem of world population, for even if we manage to control the quantity and the quality of our own numbers, unless we take a serious interest in these matters in other countries they eventually may serve to swamp us by the sheer weight of their own numbers. We must remember that thinking seriously about problems is an adaptive trait evolved during man's struggle with the environment in the effort to adjust himself to that environment, and truth and better adjustment is the success of that effort. Principally as a consequence of that effort to think seriously, man today holds the direction of his future evolution in his own hands. The knowledge we have gained of human heredity has placed a great power for good in our hands, but whether that knowledge will be used wisely and for good ends will largely depend upon two things: One is the extent to which a knowledge of heredity is understood by the people, and the other is their ability to think critically about that knowledge when it comes to its application to human and social problems. Scientists have, of course, a tremendous amount to learn about human heredity; indeed, compared to the amount that yet remains to be learned, we can truthfully say that only a beginning has yet been made in the understanding of human heredity, but that beginning, as we have seen in the preceding pages, has already been of immense practical value, and little though it is has placed the control of man's future evolution within his power.

Science and National Policy

A little knowledge, it has been said, is a dangerous thing—and so it is. The very greatest caution is necessary in dealing with all questions or recommendations which have as their object the influencing of the lives not only of the living but of those as yet unborn. There are many people, some honestly well-meaning and others dubiously so, who maintain that the restriction of immigration to “desirable types” and the “sterilization of the unfit” would greatly benefit our society. Unfortunately the voices of such individuals have sometimes made themselves so effectively heard that they have influenced national and state policies. It is perhaps not as well known as it should be that the United States Immigration Act of 1924 was based on the ill-considered, prejudiced, and unscientific judgments of the late Mr. Harry Laughlin, a worker in The Eugenics Records Office at Cold Spring Harbor, New York, supported behind the scenes by Madison Grant, the author of the notorious *The Passing of the Great Race*, and Charles Benedict Davenport, the Director of the Office, to whose remarkable judgment on matters racial in his *Race Crossing in Jamaica* reference has already been made (see page 213). Laughlin was asked to report by the House Immigration Committee in April, 1920, on the relationship of biology to immigration, and their relation to social degeneracy. Laughlin submitted his report entitled *Analysis of America's Melting Pot* in November, 1922. The burden of the report was that “the recent immigrants as a whole present a higher percentage of inborn socially inadequate qualities than do the older stocks.” On the basis of the listings in order of desirability for immigration submitted by Laughlin, both public and legislative opinion were influenced to produce an immigration act which was unsound and unjust, and which ultimately led to the absurdity of the McCarran-Walter Act of 1952 with its explicit statement that the capacity for Americanization of an individual was determined by his national or “racial” origin.*

In the light of our knowledge of human genetics and of the

* For a good discussion of this subject see Oscar Handlin's *Race and Nationality in American Life*.

knowledge we have gained from the social sciences it must be said that the national policy of the United States embodied in its quota system and legalized by its immigration acts is from every point of view biologically and socially unsound. Science, in spite of every attempt to do so, has been able to discover no connection between the biological or genetic traits of any ethnic group or nation and a greater or lesser "percentage of inborn socially inadequate qualities." Nevertheless, and in complete contradiction and disregard of the findings of science, the government of the United States has created laws which discriminate against the members of certain ethnic groups and nations who desire to immigrate to the United States.

This is but one striking example of bad genetics and worse social science written into the law of the United States. The racism implicit in the immigration laws stands in strange antithesis to the sentiments engraven at the base of the Statue of Liberty:

Give me your tired, your poor,
Your huddled masses yearning to breathe free,
The wretched refuse of your teeming shore,
Send these, the homeless, tempest-tossed, to me:
I lift my lamp beside the golden door.

Racism is contrary to all the findings of human genetics and the social sciences.

The "sterilization of the unfit" is yet another of those panaceas which are frequently urged upon us if we are to save ourselves from the alleged biological degeneration that is destined to overtake our kind unless we resort to such preventive measures. Let us quote from a model Sterilization Law drafted by the same Harry H. Laughlin to whom we have already had occasion to refer in connection with the immigration laws of the United States. In a report entitled *Eugenical Sterilization in the United States*, published in 1922, this law recommends sterilization of the "socially inadequate." A socially inadequate person is defined as "one who by his or her own effort, regardless of etiology or prognosis, fails chronically in comparison with normal persons, to maintain himself or herself as a useful member of the organized social life of the state." The sick of body and mind who are only temporarily so, and the aged sick are exempted from this definition.

We are informed: "The socially inadequate classes, regardless of etiology or prognosis, are the following: (1) Feeble-minded; (2) Insane (including the psychopathic); (3) Criminalistic (including the delinquent and wayward); (4) Epileptic; (5) Inebriate (including drug habitués); (6) Diseased (including the tuberculous, the syphilitic, the leprous, and others with chronic, infectious, and legally segregable diseases); (7) Blind (including those with seriously impaired vision); (8) Deaf (including those with seriously impaired hearing); (9) Deformed (including the crippled); and (10) Dependent (including orphans, ne'er-do-wells, the homeless, tramps, and paupers)."

The reader can judge for himself the kind of individuals who would have been directly affected had sterilization laws been in existence in their day, and also the significant number of individuals who would not have come into being at all had such laws been applied to "socially inadequate persons." One or other of the parents of many great benefactors suffered from one or other of the "socially inadequate" disorders listed by Mr. Laughlin. Had they been sterilized, humanity would have been all the poorer. As for the great men themselves, here are some of the "social inadequates" by the measure of Mr. Laughlin's definition (excluding the feeble-minded, of course):

Insane: Lucretius, Isaac Newton, Nathaniel Lee, Strindberg, van Gogh, Nietzsche

Criminalistic: Villon, Verlaine, Rimbaud, Wilde, O. Henry, Baudelaire

Epileptic: Dostoevski, Conrad, van Gogh, Caesar

Inebriate or addicted to drugs: Tennyson, Coleridge, Lamb, De Quincey, Poe, Modigliani, Dylan Thomas

Diseased: Gibbon, Rousseau, Keats, Pope, Pasteur, D. H. Lawrence, Robert Louis Stevenson, Elizabeth Barrett Browning, F. D. Roosevelt

Blind: Homer, Milton, Helen Keller

Deaf: Beethoven, Helen Keller, Edison

Deformed: Pope, Robert Hooke, Byron, Charles Steinmetz, Toulouse-Lautrec

Ne'er-do-wells: Socrates, Diogenes, Shelley, St. Augustine, Gauguin, Thoreau

Homeless: Jesus, van Gogh

Tramps: Vachel Lindsay, W. H. Davies, Walt Whitman, George Orwell

Paupers: Jesus, Gandhi, van Gogh, Francis Thompson

Some 28 states of the Union have adopted a sterilization law,

TABLE XXIX
STATES AND TERRITORIES WITH STERILIZATION LAWS, AND YEAR IN WHICH
A LAW WAS FIRST PASSED

Indiana, 1907	Montana, 1923
California, 1909	Virginia, 1924
Connecticut, 1909	Idaho, 1925
Iowa, 1911	Maine, 1925
Kansas, 1913	Minnesota, 1925
Michigan, 1913	Utah, 1925
North Dakota, 1913	Mississippi, 1928
Wisconsin, 1913	Arizona, 1929
Nebraska, 1915	West Virginia, 1929
New Hampshire, 1917	Oklahoma, 1931
Oregon, 1917	Vermont, 1931
South Dakota, 1917	South Carolina, 1935
Alabama, 1919*	Georgia, 1937
North Carolina, 1919	Puerto Rico, 1937
Delaware, 1923	

*Law has been inoperative since 1935 when State Supreme Court rendered an adverse opinion regarding broader sterilization legislation then pending.

but in most states the law has only been desultorily applied. The states having such laws on their books and the year of their enactment into law are shown in Table XXIX. Those who believe that sterilization would significantly reduce the number of "the unfit" usually operate on the naive assumption that every individual sterilized would by so much, at least, reduce the frequency of the unfit. But this is not the case at all. Sterilization is not an effective or quick way of eradicating hereditary diseases or abnormalities. For example, in a population such as that of any large city in which random mating is the rule, let us suppose that 1 percent of the members of that population were affected by a simple recessive abnormal condition. Now let us suppose that all these individuals were sterilized. It would require four generations, a hundred years, to reduce their number to 0.5 percent, seven generations or from 175 to 200 years to reduce their number to 0.25 percent. If sterilization were initiated with a disease due to a simple recessive with an incidence of 0.5 percent, it would take

six generations or 150 years to reduce its frequency. The reason for this is that a large number of individuals carrying the gene in its heterozygous recessive state would not exhibit the defect associated with it but would transmit it. In addition, mutant genes would add to the number of individuals, born of apparently normal parents, who were affected.

Sterilizing the healthy relatives of the diseased individuals might serve to accelerate the reduction in the frequency of the abnormality, and of course would also reduce the frequency of the normal offspring that would otherwise have been born to such individuals, whether they were carriers of the defective gene or not.

One percent of the general population is believed to be affected by schizophrenic tendencies. It is considered that about 10 percent of the children of schizophrenics are likely to become schizophrenic. This means that 90 percent of the children of persons who develop schizophrenia will be normal. Does anyone have a right to sacrifice the 90 percent normals in order to prevent the 10 percent of abnormals from coming into being? * I think that there can be no doubt that no one has such a right nor should anyone ever be granted such a right. To remain with the example of schizophrenia, there is today good reason to believe that this dreadful disease is already becoming amenable to the newer treatments in psychiatry, and there is some promise that cures on a much larger scale than are possible at the present time may be effected in the not too distant future. Scientific research and not surgery is the proper approach to the problem of schizophrenia, as it is to every other hereditary disorder.

There are relatively few disorders which, affecting both parents or the genes for which are carried by them in their germ cells, are capable of affecting *all* the children born to them. As we have already seen in such a hereditary disorder as hemophilia, in which nearly every individual suffering from it used to die before

* In point of fact, the total reproduction of schizophrenics is considerably lower than that of any other comparable population. Severely affected schizophrenics are much less likely to produce children than the less severely affected, and as Kallmann has pointed out in *Heredity in Health and Mental Disorder*, "a schizophrenic who has children is apt to have relatively mild symptoms and therefore a comparatively high degree of resistance to the disease. The factors producing this resistance will be passed on to the children. Where both parents are schizophrenic the children will inherit this capacity for resistance from both parents."

reaching adult years, while it is today not possible to cure the disorder, and it may never be possible to cure hemophilia, nevertheless it is possible to control acute bleeding episodes, to relieve the sufferer of much pain and discomfort, and to enable him to carry on his work fairly normally. There is great promise that there will soon be a major breakthrough in the treatment of hemophilia, so that it will be possible to help hemophiliacs precisely as it is today possible to help diabetics. Once the hemophiliac reaches adult years, which he does in most cases owing to the progress of modern medicine, he is able to reproduce. In so doing he increases the frequency of the number of individuals who will suffer from hemophilia. The question then arises, Is medicine doing the right thing by multiplying the possibilities for increasing the number of persons who will suffer from hemophilia?

That is an important question, and so is the answer to it. The answer is that medicine, while increasing the possibility of the number of individuals who will suffer from hemophilia, is by the very same means increasing the possibility that hemophilia will become a much less serious disability, for the very means that makes it possible for hemophiliacs to achieve adult years is the same means that will make it possible for them to live fruitful and happy lives—which large numbers are doing today. There are several hemophilic physicians working today in the field of hemophilia who have already advanced our knowledge of the treatment of that condition.

For the sake of those who are dubious about the benefits of sterilization as well as for the sake of those who are not, let me quote the case of a twenty-two-year-old Argentine hemophiliac reported by Dr. A. Pavlovsky who has attended him since his birth. Dr. Pavlovsky shall present the case in his own words:

There is no record in his family history of hemophilia until his generation: 3 hemophilic brothers and a sister carrier. The sister married and had 3 children, 2 hemophilic males and a daughter (carrier). He presents one of the severest forms of hemophilia I have ever seen. He has been on various occasions at death's door. For long periods of time this boy was bed-ridden and suffered severe pain during his many hemorrhagic crises. Mercifully today his disease has become less severe, as is generally the case when a hemophiliac reaches adult age. The only sequelae that remain are only slight ankylosis of both ankles and elbows. Always an outstanding pupil, this patient is

now studying Law. He is a well-known chess player, and represented the Argentine in the "International Tournament for World Championship" in England. He has many interests in his life, amongst others, one that deserves mention. He uses his free time at week ends to visit lepers to whom he teaches chess. While the Argentine was under the dictator's control this boy gave an example of patriotism and courage. For 5 months he was imprisoned for defending his democratic ideals, and when freed he was immediately gaoled again for protecting the Cathedral of Buenos Aires against the mob sent to sack and burn it. On his release from his second term of imprisonment he at once volunteered to carry bombs and firearms for the revolutionaries. When asked his opinion this young hemophiliac said that in spite of his sufferings; his parent's anxieties; the limitations of his own life; the constant uncertainty of what the morrow might bring forth, he was glad to be alive and to be able to help others. He added that should he marry, he would have children, as he considers that they should be able to bear what he has borne.*

Either the normal gene *H* had mutated to the abnormal gene *h* in this young man's mother or she was a carrier of the gene inherited in the usual way. Had her carrier state been detected, should she have been sterilized? I think the answer is clearly no. The whole world would have been the poorer had this young Argentine hemophiliac never come into being. Indeed, the world would be a far happier place if it were endowed with more persons of his character, no matter what hereditary disorders they suffered from, rather than millions of physically healthy non-descripts!

The Mentally Defective

In the scale of humanity every human being has a value, whatever his qualities may be, and unless he is a complete idiot, even if he is feeble-minded, as long as he is able to work he can be a useful member of society. As the great geneticist J. B. S. Haldane has said: "I am of the opinion that a man who can look after pigs or do any other steady work has a value to society, and that we have no right whatever to prevent him from reproducing his like." Indeed, many farmers prefer feeble-minded men to look

* A. Pavlovsky, "The marriage problem and descendants of hemophiliacs," in *Hemophilia and Hemophiloid Diseases*, edited by Kenneth M. Brinkhous.

after their pigs. Another great authority on the genetics of the mentally deficient, Professor L. S. Penrose, writes in his book *Mental Defect*: "A striking feature of defectives of imbecile and lower . . . grades is their apparent incapacity for being bored with an occupation; and provided some simple manipulation can be taught, the defective is perfectly happy in continuing the same manipulation for days and years without any change. This fact makes possible methods of dealing with patients who might otherwise be difficult to employ. In a regular, even if very monotonous, employment they learn to be useful and worthy people."

Clearly mental defect is not so much a biological as a social problem, for in a society in which occupational status would be measured by ability, places could be found for persons of very limited ability. To a large extent our society already does this, for the number of persons who are something less than bright among us runs into several millions. It is estimated that at the present time there are well over 2,000,000 mental defectives in the United States. There are also at the present time some twenty-eight states scattered throughout the country that have laws permitting the sterilization of mental defectives. Over 60,000 individuals have been sterilized since the first of these laws was passed in 1907.

Mental defectives do not reproduce themselves efficiently. The idiots, those who belong to the lowest grade of feeble-minded, and are unable to do anything for themselves, have a mortality rate which is more than five times greater than that of the general population, and in the younger ages their death rates are nearly ten times greater than that of the general population. The imbeciles, who can learn simple manual tasks, have a death rate that is about twice as high as that of the general population. Among morons, who have a mental capacity reaching to the borderline of low normal intelligence, death rates are also higher at every age than for the normal population. The greater the degree of feeble-mindedness among the mentally defective, the lower is their reproductivity, so that a natural limiting effect is operative upon their multiplication.

The great majority of mental defectives are born to perfectly normal parents; hence, the sterilization of the mentally defective would scarcely result in a significant reduction in the number of mental defectives born.

The truth is that the good will to help is blind and often cruel if it is not guided by true insight based on knowledge. A physician can only be of genuine use when the disease for which he is prescribing has first been carefully investigated. Only then can he be sure what his remedies can effect; otherwise he is a positive danger. Voluntary or compulsory sterilization of "the unfit" is an ill-conceived prescription by physicians with too little knowledge of the disorder for which they are prescribing.

Every so often some self-appointed pundit, often speaking with the authority of some institutional affiliation which renders him in his own opinion and that of the public an expert on the things he is least qualified to judge, delivers himself on the subject of "man, the mechanical misfit." Man's upright posture is blamed for his pendulous abdomen and his back troubles, while the obvious fact that man's pendulous abdomen may in large part be due to his poor habits of posture, eating, and exercise, and his back troubles be due to the beds he sleeps in, and the chairs and automobile seats he spends so much of his life in, are altogether overlooked. Such authorities also proceed to inform us from time to time that man's social troubles are principally due to his biological deterioration, to the carelessness with which he allows the defective to reproduce and pollute the germ plasm of the species.

It is of course true that there are many individuals among us who are genetically more or less seriously defective and who are capable of transmitting their defects to a certain proportion of their descendants. In the case of rare recessive defects such as infantile amaurotic idiocy, nothing has to be done since these conditions in large populations eliminate themselves by the early death of the victims. In the extreme cases such as idiocy, social regulation is necessary, not surgical intervention. Surely, no one will *seriously* suggest that such individuals are responsible for our social ills. Our troubles emanate not from biological idiots but from social ones; and social idiots are produced by society, not by genes. It is, therefore, social and not biological therapy that is indicated.

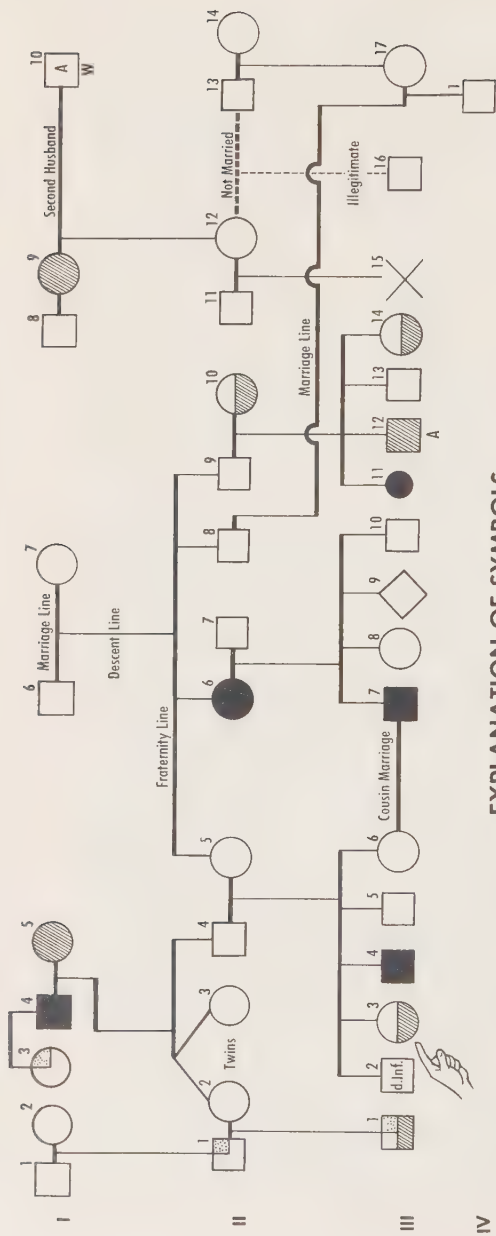
The Problem of Defectives

Owing to the fact that a certain proportion of normal genes mutate in every generation to the abnormal form, genetically

transmissible defective genes will always be coming into being afresh in every human society—but so will the fresh knowledge which will enable us to deal with them. At the present time we are rapidly developing new methods of detecting carriers of defective genes even before those genes have a chance to be transmitted or to express themselves. For example, deficiency in Ac-globulin, or clotting factor, can be picked up by an analysis of the blood of the affected individual. Such a deficiency is due to a recessive autosomal gene. In the homozygous condition the defective gene produces the deficiency disease, parahemophilia. Thus, by analysis of the blood of potential parents who are both carrying the gene it would be possible to tell them what the effects are likely to be upon their children if they decide to marry and raise a family. It is similarly possible to do this for many hemophilia-like conditions as well as several other unrelated conditions such as possible erythroblastosis in an infant of Rh-incompatible parents, A-B-O incompatibilities, and the like.

These are considerable achievements, we have every reason to be confident that future advances in genetics will put the control of human heredity even more efficiently within our power. Even the control of mutations is today being discussed by geneticists as a practical possibility. We can, therefore, look to the immediate future hopefully and for the present realize that there is nothing that such severe measures as sterilization can do that soundly based social control cannot do a great deal better in every way. There is no substitute for sound knowledge humanely applied.

All of us are carriers of some deleterious genes. Such genes are usually recessive. Under a system of random mating such as exists in most civilized countries the accident of marriage sometimes brings such recessive genes together, which will then express themselves, to some degree, in some of the offspring issuing from such a union. Where deleterious genes are carried in the recessive state, there is usually a history in the family of the expression of the gene—in a grandparent, or uncle or aunt, and in some of their offspring. All of us ought to draw up a pedigree of our families, as far back as we can, and extend it laterally as far as we are able. It is a simple matter to prepare such a pedigree. A sample pedigree is shown in figure 42. You can draw up separate pedigrees of the same family for separate traits or you can put them all into one pedigree. Apart from being fun and instructive, such pedigrees ought to be as obligatory as a birth certificate or a marriage li-



EXPLANATION OF SYMBOLS

(a) Standard for all pedigree charts:

□ = Male; ○ = Female; ◇ = Sex unknown; ● = Still-born or Miscarriage × = Children Number and sex unknown; d.Inf. = Died in infancy; = Parents. = Children.

Roman figures to the left indicate generations; arabic figures locate individuals (thus III,7 is the young man in the third generation who married his cousin). Offspring should be listed in the order of birth; for example, in generation III on the chart the four siblings of the propositus are first a brother, then two brothers and a sister in the order of birth in relation to the propositus in which they occurred.

The following letters, placed in or around the individual's pedigree symbol, are standard for certain traits: A-alcoholic; B-blind; D-deaf; E-epileptic; F-feebleminded; I-insane; M-migraine; N-normal in reference to traits under consideration; Ne-neurotic; P-paralytic; S-syphilitic; T-tuberculous.

(b) To fit the particular family and traits (whether physical, mental, or temperamental, good or bad) under consideration, invent special symbols, or select special letters or letters in combination with numbers (in addition to those standardized above) to be placed within or near the particular individual's pedigree symbol, to indicate particular traits and their degree of development.

FIG. 42. Pedigree Chart Showing the Manner of Construction and Standard and Special Symbols.

cense. A couple contemplating marriage could go over their pedigree charts and from them discover whether they stand in need of genetic counsel. In any event, they could, on a purely voluntary basis, take their charts to a genetic counselor,* who could then advise them. While human beings continue to select their mates for reasons which seldom include a mutual inspection of their genes, it is foolish to talk of eugenics, or selective breeding from the best stocks. Human beings will continue to breed at random for a long time yet, and it is perhaps just as well that they do. And if under this system of random mating, which has served humanity so well in the past, some intelligent individuals marry some feeble-minded ones, it is a real question whether in the long run many of the genes carried by the feeble-minded may not be just as valuable to the human race as those carried by the normally intelligent.

We must take the world as we find it and do what we can to leave it a better and a happier place than we found it. Toward this end the most important role that the individual can play in any society is that of a parent. The creation of a good human being is the greatest contribution that the individual can make to his society. And there can be nothing more gratifying to the individual than the making of such a contribution. This being so, the first consideration of the potential maker of a human being should be not what the biological characteristics of his offspring are going to be, but whether he, the potential parent, is prepared to do his best to make the lives of his offspring as useful and happy as possible, whatever the biological characteristics of his offspring may be. In the final analysis I think this is the long and the short of it.

This means that potential parents who are fully aware of the fact that they will almost certainly bring a certain number of children into the world who will suffer from certain hereditarily determined handicaps should be perfectly free to do so, if they thoroughly understand what they are about and will always be ready to do what needs to be done to help their handicapped children realize their lives as fully as possible. But where the potential parents know themselves well enough to understand that their own personalities are such that they would prove far greater handicaps to a happy family life than any genetic handicaps that

* Genetic counseling services are available in several of our hospitals and at clinics attached to some of our universities. See Appendix C.

their children would have, it might be the path of wisdom for them to desist from having children.

In his excellent little book *Counseling in Medical Genetics*, the director of the Dight Institute for Human Genetics at the University of Minnesota, Dr. Sheldon Reed, writes, "Our serious clients come to us because they are troubled. They show great affection for their abnormal child and give it more than its ordinary share of attention, but the parents are unhappy both for the defective child and for themselves. We have never seen parents who wished to repeat the misfortune." There can be little doubt that in the vast majority of cases this is true, and that most parents will wish to avoid bringing defective children into the world, not because of the genetic hazards to the race but because they are themselves rendered unhappy by the presence of such children. This is, however, not always the case. I know of one husband and wife of more than average intelligence who in spite of the fact that their first-born was a hydrocephalic, the second-born a normal, and the third-born a mongoloid, are still bent on having children, for they have found both the hydrocephalic and the mongoloid very lovable indeed, and the whole family is a happy one. But this is the exceptional case. I see, however, no good reason why it should not become more frequently the case that parents with defective children are able to accept the condition of their children as a challenge to do the best that they can for them, and to be quite happy doing so. The matter, however, is by no means as simple as all that, for as Dr. Bernard Farber has shown in a recent study, the presence of a severely mentally retarded child, especially a boy, can have a seriously disrupting effect upon marital relationships and adversely affect the personality development of the normal siblings. These are considerations which all parents faced with the possibility of a retarded or otherwise defective child must be prepared to give their most careful attention.

Old-fashioned ideas and artificial standards often conspire to make the parent of the defective child feel ashamed, even guilty, as if some offense or solecism has been committed. But in the light of modern genetics and medicine it is today fully understood that such defective children could occur in any family, and that no matter what family they have been born into, they should be no more an occasion for shame or guilt than the development of pneumonia or poliomyelitis in ourselves. Both diseases are avoid-

able ones, but a great deal of luck affects the avoidability, and so it is with the birth of defective children. What is called for is understanding and the proper treatment. A good environment can make up for many of the defects of the germ plasm.

It is not simply a knowledge of human genetics that is necessary but also a knowledge of human nature, especially self-knowledge, when we come to deal with the behavior of ourselves or of other human beings. But a knowledge of genetics, and especially of our own genetic history, is immensely valuable in assisting us to determine what our behavior should be, if not in relation to marriage, that is to whom we marry, then in relation to whether or not to have children. In this connection one of the most frequent questions that comes up is the matter of *consanguinity*, that is, whether or not to marry a "blood relation." As I have pointed out earlier in this book, blood has nothing whatever to do with determining genetic relationship. What the question "Shall I marry my cousin?" in fact means is, "Shall I marry an individual whose genes are in part derived from the same source as one of my parents?"

Since there are at least twenty-four states in the Union that forbid the marriage of first cousins, the question is a legal as well as a genetic one, involving the legal status of offspring, their right to inherit possessions, and their inheritance of possibly deleterious genes.

On this subject there has been a great deal of loose thinking. Yet the facts are quite simple. If the marrying partners come from a good healthy stock, that is to say, one in which no significantly disturbing disorders have been noted, then the chances are high that the offspring will not exhibit any such disorders. We have pedigrees of many distinguished families which bear abundant testimony to this fact. Brother-sister marriage was not uncommon in ancient Egypt and was the rule among the Pharaohs. A prince was held to have an unquestionable right to the throne only if he was born of parents who each had equal rights to the throne. By the beginning of the Eighteenth Dynasty (about 1580-1350 B.C.), when brother-sister marriage had already been long in practice, there appeared a succession of the most brilliantly gifted rulers Egypt had ever known. A few of the royal personages involved are shown in the following pedigree.

It was Aahmes I, and his sister-wife Aahmes-Nefertari, who ex-

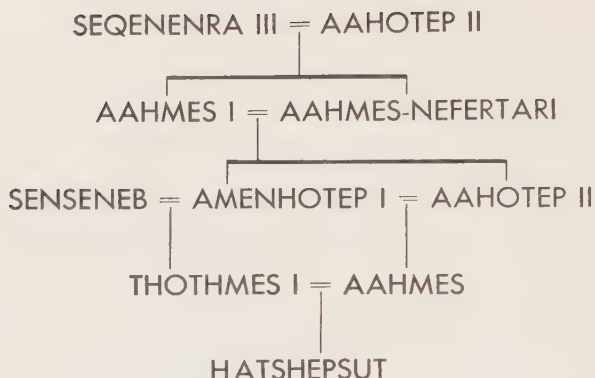


FIG. 43. Portion of the Pedigree of Egyptian Rulers Illustrating Their Close Inbreeding Practices.

pelled the Hyksos and founded the Eighteenth Dynasty. Aahmes-Nefertari was later deified. Perhaps the most remarkable in the line was Queen Hatshepsut, whose father and mother were half brother and sister, her mother Aahmes being the result of two successive marriages between full brothers and sisters. Hatshepsut's husband, Thothmes III, is believed to have been a cousin of the queen.

The Ptolemies, who ruled Egypt from 323 to 30 B.C., usually married their sisters. They are sometimes quoted as a good example of the effects of inbreeding. Upon this score there are two things to be said. One is that the Ptolemies were a mediocre lot to begin with, and the second is that they were by no means as inbred as is sometimes alleged. Finally, the famous Cleopatra (69–30 B.C.), the last of the line, was not the offspring of a brother-sister marriage as is frequently stated, but of her father Ptolemy XI and an unknown woman.

While a violent lot—the first Ptolemy was a general in the army of Alexander the Great—the Ptolemies were not particularly known to suffer from any physical or mental abnormalities.

Before the advent of Moses, the Jews married close relatives by preference. Abraham married his half sister, Sarah (Genesis xx.), and the mother of Moses was his father's paternal aunt (Exodus vi. 20). The Mosaic codification of the laws prohibited all such unions. One cannot help wondering why.

With the beginning of the Christian Era, the pronounced objection to the marriage of near kin appears to have been based on the ground that such marriages tend to produce defects in the offspring. In 1215 A.D. the Church finally decreed that only persons related beyond the degree of third cousins might marry, and this is essentially the law of the Church today.

In general the prohibition placed on the marriage of close relatives is well founded, because in the large populations of the civilized world there are distributed many recessive genes for defective traits. It has been estimated that in such populations every individual carries about eight of this large pool of recessive genes. It should be obvious that individuals who are close relatives and derive many of their genes from the same sources are much more likely to contribute a matched pair of such recessive genes to their offspring than unrelated individuals. Hence, fairly rare hereditary disorders would be likely to occur more frequently in the offspring of related individuals than in unrelated—and this is found to be the case. This is illustrated in Table XXX in which an extremely high frequency of first-cousin marriages is shown among the parents of individuals suffering from rare diseases due to recessive genes. Indeed, we have learned that when a rare hereditary disorder appears in an individual it is quite probably going to be found that the parents are close relatives such as first cousins. The

TABLE XXX

EXTREMELY HIGH RATE OF FIRST-COUSIN MARRIAGES AMONG THE
PARENTS OF CHILDREN WITH SOME RARE HEREDITARY DISORDERS

Disorder	Percent First-Cousin Marriages Among Parents
Pseudohermaphroditism	37
Alkaptonuria	33
Xeroderma pigmentosum	26
Congenital ichthyosis	24
Albinism	17
Infantile amaurotic idiocy	15
Total color blindness	11
Friedrich's ataxia	10

chance that a cousin carries the same recessive gene, say for albinism, in first-cousin marriage is about 1 in 8,* as compared with 1 in 70 unrelated individuals, so it will be seen that first-cousin marriages are quite a hazard. This should not be surprising in view of the fact that having two grandparents in common, first cousins therefore share more than one eighth of their genes in common.

In the United States and in England about one half of 1 percent of marriages are contracted between first cousins, but in many localities of the world which are small and in which the populations are isolated either geographically or socially or both, the percent of marriages between first cousins may be very high. In many nonliterate societies it is obligatory to marry one's first cousin; in still others one marries one's cousin a generation removed. Such communities show the expected results of cousin marriages: many of them are distinguished by a marked absence of hereditary defects, while others show a marked presence of such defects. For example, the famous Pitcairn Islanders, the Batz community of Loire-Inférieure, the Hindu community of the Tengger Hills, Java, and the inbred populations of numerous small islands appear to show no ill effects of such inbreeding. On the other hand, ill effects have occurred in such communities as that of the Nanticoke Indians of Delaware in such physical traits as superior palpebral ptosis (drooping of upper eyelid), the population of Martha's Vineyard in the frequency of deafness which runs in a number of families, the hill folk of New England with the prevalence of feeble-mindedness, and the toothless men of the Amil community of Hyderabad in the province of Sind, Pakistan, where within six generations many families have appeared whose members are the descendants of a single individual with regard to a sex-linked recessive gene responsible for a large number of the males being entirely toothless and without sweat glands.

Clearly it is not inbreeding, no matter how close the inbreeding may be, that is responsible for the appearance of defective traits. Whatever effect inbreeding may have is due entirely to the inheritance received. If that inheritance is good the effect will be good; if it is bad the effect will be bad; if it is indifferent the effect will be indifferent. In this connection we cannot do better than

* For the method by which this figure is arrived at see Appendix B.

quote the words from East and Jones' classic book *Inbreeding and Outbreeding* (page 244):

Owing to the existence of serious recessive traits there is objection to indiscriminate, irrational, intensive inbreeding in man; yet inbreeding is the surest means of establishing families which as a whole are of high value to the community. On the other hand, owing to the complex nature of the mental traits of the highest type, the brightest examples of inherent ability have come and will come from chance mating in the general population, the common people so-called, because of the variability there existent. There can be no permanent aristocracy of brains, because families, no matter how inbred, will remain variable while in existence and will persist but a comparatively short time as close-bred strains. But he is a trifler with little thought of his duty to the state or to himself, who, having ability as a personal endowment, does not scan with care the genealogical record of the family into which he enters.

And that will remain at once the best statement of the facts and the best advice on marriage that can be given. Marriage with a relative is a risk, but it should be a calculated risk. For that matter all marriage is a risk, but it is much more of a risk, genetically speaking, when it is contracted between relatives because one can rarely be certain of the absence of deleterious recessive genes carried in the heterozygous state by each partner. When Charles Darwin married his cousin Emma Wedgwood in 1839 he could have been fairly certain, looking back upon the distinguished backgrounds of both his own and his wife's family, that the children he and his wife would have would be reasonably satisfactory examples of humanity. They were indeed. Darwin's sons became distinguished scientists and leaders of thought in their own right, as have many of his grandchildren, both male and female. (See fig. 44.) If there is reason to believe that the inheritance to be passed on to one's offspring in a marriage between relatives is likely to be free of deleterious influences, such judgment being based on a knowledge of the genealogical record on both sides, the genetic risk would be very slight. On the other hand, where the genealogical history on even one side points to the presence of unfavorable recessive genes, it would be wise, if such a union is entered into, to do so with a full understanding of the possible consequences if there are to be any children.

The question whether anyone has the right knowingly to

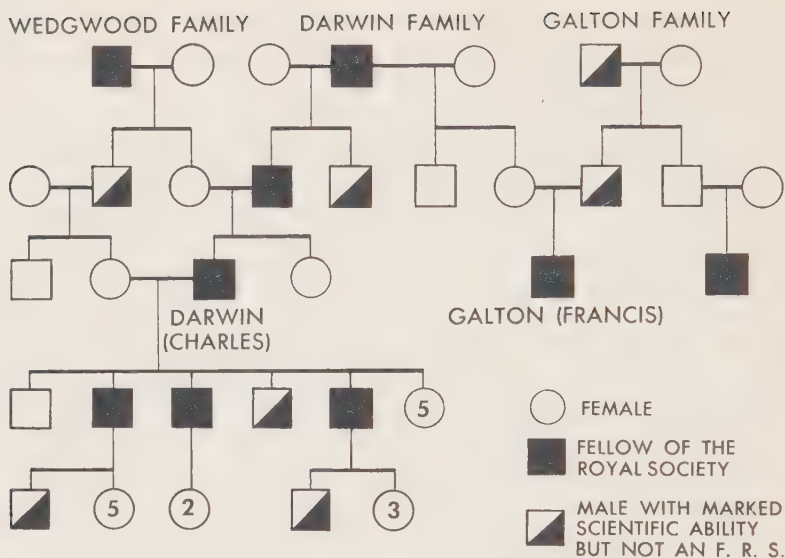


FIG. 44. A Short Pedigree of the Darwin Family.

bring children suffering from a hereditary defect into the world is not an easy one to answer. There can be no question that infantile amaurotic family idiocy is a disorder that no one has a right to visit upon a small infant. Persons carrying this gene, if they marry, should never have children, and should, if they desire children, adopt them. But what shall we say of such conditions as total color blindness or albinism? Shall persons carrying the genes for these conditions also desist from having children? I think the answer to that question would be best left to the decision of the potential parents themselves, provided they fully understand the meaning of their decision. It seems to me that what is required is not sterilization or legislation but a more widespread understanding of the genetic facts and the human responsibilities involved, not only by parents but by the larger society.

A society does not properly acquit itself of its responsibilities to its citizens by either choosing to ignore the existence of defectives and placing the entire burden of their care upon the parents, or else by approaching the problem with the crude and ineffectual scalpel of sterilization. Our society should make

much more varied and substantial assistance available than it does at present to the family with one or more defective children. And to forestall the advent of undesired defective children, genetic counseling should be available premaritally, and is the most significant clarification that ought to be interestedly sought by the young couple contemplating marriage. The best time and place to prepare individuals in such knowledge-seeking attitudes is in the schools, for an elementary knowledge of the facts of genetics, of human heredity, should become part of the intellectual equipment of every ordinarily educated citizen.

That aspect of eugenics which suggests the negative control of the multiplication of undesirable hereditary traits is, in general, misconceived, for the following good reasons: (1) Most of the more serious disorders are self-limiting since their bearers generally do not propagate; (2) many hereditary disorders are amenable to environmental alleviation; (3) the increase in human mobility and the collapse of innumerable barriers to intermixture between large numbers of the members of populations that were hitherto separated reduces the chances of deleterious recessive genes coming together; and (4) sterilizing homozygotes who show a defective trait due to recessive genes still leaves by far the greater number of individuals carrying the gene in the heterozygous state to circulate it freely throughout the population. If, for example, the frequency of a recessive gene in the general population were 1 in 1,000, that would mean that the homozygote in whom the defective trait was expressed would occur in 1 out of 1,000,000 individuals. Supposing the homozygous individual were sterilized, that would still leave 999 heterozygotes to distribute the gene. Clearly a rather inefficient and ineffectual way to deal with the problem. Finally, (5) since the number of genes of any given sort in the human species is usually very large, any artificially induced changes in their number in any local portion of the species is likely to have very little if any effect upon the total frequency. As Penrose has put it, "The wider problem of genetical improvement of the human race must be viewed against the background of gene frequencies in the world population and the relative fitnesses of different phenotypes in different environments."

Positive eugenics has somewhat more to be said in its favor insofar as it seeks to underscore the necessity of paying more at-

tention than we do on the one hand to the choice of mates and on the other to the greater encouragement of reproduction of the well-endowed. Unfortunately, eugenists in the past have placed the emphasis almost exclusively on intellectual qualities as the principal criterion of the well-endowed. Intellectual qualities are important, but so are other qualities of mind, such as adaptability, integrity, compassion, and balanced temperament. Health may be defined as the ability of the organism to work efficiently and to love—to confer survival benefits upon others in a creatively enlarging manner. I think it can be successfully argued that these traits are ultimately more important than the exclusively intellectual ones for the survival and healthy growth and development of the individual and the species. Intellectual qualities and special abilities are, of course, of the highest value, but they do not constitute all that it takes to make a complete human being or a society. Human beings are not ants, nor should they try to imitate them in spite of Solomon's injunction, which was simply his way of commending industry. In any event, we don't by any means know enough about man's genetic system to commence modifying it by special types of breeding. Domestic plants and animals having a commercial value may be bred for special traits which improve their desirability, but as every breeder knows, in developing such desirable traits many undesirable ones may turn up and the strains or the specimens exhibiting these undesirable traits have to be discarded.* It is scarcely possible or desirable to follow such a procedure in the case of man. Desirable traits in man, insofar as they are affected by gene differences, appear to be the result of the interaction of many genes, and there is every reason to believe that the infusion of new genes into a stock is, on the whole, to its advantage, so that breeding in the open system of random mating is likely to be of greater benefit to the species than is breeding which is directed toward the development of some special trait.

* For example, hornlessness is a valuable trait in goats. In breeding for hornlessness, the breeders have unwittingly increased the frequency of the gene for pseudohermaphroditism or intersexuality. The dominant genes for hornlessness and the recessive genes associated with intersexuality are somehow linked. The recessive genes under normal conditions remain unexpressed, but under controlled breeding conditions find expression as a result of their unsuspected linkage with the genes associated with the trait considered desirable. Since the intersexual goats, who are reversed genetic females, are sterile, the breeders are introducing a much more undesirable trait into the population of goats than the one they sought to eliminate.

We know that musical ability, mathematical ability, and the ability to paint are closely associated with certain genes. These seem to be few in number. It would be possible to build up whole families of individuals with such special abilities. The Bachs and the Mozart-Weber families are examples of musical dynasties that did this for themselves. The Bernoullis are an example of a mathematical family of great genius; the Pissarros, Camille and Lucien, are an instance of father and son who were great painters. Anyone interested in creating a line of great musicians, mathematicians, or painters, who does not himself possess any of these abilities had better look for a mate who comes from a family that does.

Planned breeding can be left to the individual. I do not think that the State should do anything other than make it possible for the individual to acquire all the knowledge he needs to make the best of his own heredity and to do what he ought and can about the heredity of his descendants—remembering, always, that whatever the genetic inheritance of the individual is, one can do a great deal through the agency of the environment to improve its expression.

The Bomb, Radiation, and Human Heredity

THE ATOM and hydrogen bombs brought to the attention of the whole world the fact that Western man now had in his possession a means of bringing death and disaster to vast numbers of human beings in a matter of moments. The progressive increase in the destructive power of the bombs that have since been manufactured, the hydrogen bomb tests and their accompanying fallout, have increasingly served to focus attention upon the dangers to the human species of misused atomic energy.

The constant threat of nuclear war has further served to concentrate attention on the annihilative effects upon human beings and cities, not to mention the sufferings of those who managed to survive. It has been said that in a nuclear war the use of a sufficient number of atomic weapons could physically put an end to the whole of the human species. This is theoretically possible, but practically it would be extremely difficult. Civilized man might certainly succeed in physically wiping himself off the face of the earth, but there are many people in far-removed places of the earth who would certainly not be so eliminated. However, their eventual elimination would come about quite as certainly through the long-term effects of the radiation to which they had been exposed, assuming that this was quantitatively and qualitatively significant.

Let us hope that no individual and no nation will ever be so demoted as to use such suicidal devices for destructive purposes.

Meanwhile, the very fact that such devices are being created in large numbers, that their testing releases dangerous by-products, and that the waste products of atomic energy plants present serious disposal problems, all constitute conditions which in themselves may seriously affect the immediate or long-term well-being of the human species. The damage done to the hereditary structure of the human species by the energy released from such artificially created sources of radioactivity may in the long run prove far more effective in putting an end to *Homo sapiens* than an outright nuclear war. This is the extreme possibility—the end of the human species, as a consequence of man being just a little bit too clever for his own good. Anything less than this extreme possibility amounts to such an increase in the number of hereditarily defective individuals of every sort, that in terms of the human tragedies involved, it is utterly indefensible for *anyone* ever to permit the use of any device that could produce such avoidable suffering. We are here concerned with matters of life and death, not only as they affect the lives of those now living but also as they will affect the lives of generations of our descendants, of our fellow human beings, who are to follow us from now on into the centuries and the millennia. We of the twentieth century have a grave responsibility placed squarely upon our shoulders, and it is therefore desirable that we make ourselves as fully acquainted with the facts as we are able, for what we do or do not do about them will largely determine the future of ourselves and of our descendants.

Radiation and Mutation

Radiation is the process in which energy, in the form of rays, waves, particles, beams of particles, or heat, moves from one place to another—it is energy on the move. We feel the radiation from the sun in the form of heat, and observe its effects in ourselves after more or less prolonged exposure to it on the beach. Getting too hot from the rays of the sun can make us very uncomfortable and even cause us to be sick. Getting burned from the rays of the sun can be pretty serious, and cancer of the skin of the face as a result of overexposure to the sun is a well-known consequence of its disordering effects. These are the kinds of disordering effects of radiation that you can feel and see. By far

the most sensitive system in the body to the effects of radiation is the inheritance system, the reproductive cells, the carriers of the hereditary materials, in the gonads, in the ovaries of the female and the testes of the male. Any radiation reaching the reproductive cells causes mutations (changes in the material bases of heredity), and such mutations are passed on to succeeding generations. Over 99 percent of mutations are harmful. Thus, any increase in the amount of radiation which human beings normally receive from natural sources endangers the adaptive equilibrium attained by the human species in the course of evolution by producing an imbalance as a consequence of the greater number and kinds of mutations which the species is suddenly called upon to support.

All matter is made up of atoms. An atom is composed of a nucleus surrounded by a swarm of electrons. The nucleus is composed of protons and neutrons. Electrons are very light, fast-moving particles carrying a negative electric charge. They are sometimes called *beta rays*. Protons are about 2,000 times as heavy as electrons and are positively charged. Neutrons are about the same size as protons, but are uncharged.

Alpha particles, made up of two protons and two neutrons, are the nuclei of helium atoms. They are swiftly moving particles of high energy, carrying a positive electric charge but with little power of penetration.

Every element has a unique and definite number of protons. The atomic weight of an element is determined by the weight of its protons and neutrons. Two atoms having the same number of protons but different numbers of neutrons are known as *isotopes* of the same element. Isotopes are today artificially produced in great numbers and are proving of the greatest value in experimental medicine, although care in their use is now more than ever indicated.

When atoms get overenergetic or "excited," they radiate, that is, emit, the various particles of which they are constituted—those named above as well as others.

An atom gets excited when it is knocked about, as it were. When you throw atomic particles against other particles in the nuclei of atoms, as in an atom-smashing machine, the target nuclei get excited and emit at great speed a great variety of particles, including gamma rays. *Gamma rays* are electromagnetic

radiations of extremely high energy, having great penetrating power which can traverse the whole body with relatively little absorption. *X rays* are very similar to gamma rays and are produced by high-energy electrons. X rays are usually produced artificially in special electrical machines, in which a stream of fast-moving electrons is made to strike a metal target. As a result some of the atoms in the target get excited and start emitting some of the energy they have acquired from the electrons that have been hitting them, sending out their own electrons in the form of X rays. The number of electrons sent out will vary in their penetrating power according to the electrical energy used in their production. The biological effects of X rays are brought about by the high-energy electrons that are liberated in the tissues.

All these types of radiation are known as *ionizing radiations* because in their passage through anything they leave electrically charged atoms and molecules, called *ions*. When an atom is hit by or collides with other atoms or atomic particles one or more electrons are either gained or lost as a result of the impact.* Such gain or loss occurs during chemical reactions in which electrons are transferred from one atom to another by the action of radiant energy of one kind or another. It is this process of electron detachment from atoms and reattachment to other atoms that is called *ionization*. The physicochemical changes produced in living tissues which results in radiation damage, in the form of the death of cells or altered heredity, are due to the changes initiated by the ionization process. The efficiency of a given dose of radiation in producing biological changes can be related to the number of ions that are produced per unit length of the track through which they pass.

All ionizing radiations, however low the dosage, are capable of producing mutations.

There are some substances which are naturally radioactive, such as radium or uranium. They are not dependent for their radioactivity upon the collision of atoms or their particles, but their atoms are just naturally in a state of excitement. From time to time their nuclei erupt, giving off alpha particles, electrons, or gamma rays.

* The loss of electrons results in a positively charged ion, called *cation*; the gain of electrons in a negatively charged ion, *anion*.

In addition to naturally radioactive atoms and atoms that are made radioactive by bombardment, there is yet another important natural source of radiation. This is outer space, from which high-energy particles called *cosmic rays* shower upon the earth. It is believed that these cosmic rays have been the principal cause of mutation in all living things.

The intensity or dosage of radiation which the living body receives can be determined by measuring the ionizations produced. The unit of dosage is known as the *roentgen*,* usually abbreviated to the simple letter *r*. In the human body an *r* corresponds to about 2 ionizations per cubic micron, a micron being $\frac{1}{25,000}$ of an inch. An exposure of one *r* of radiation over the whole body—which is a small amount of radiation—will result in 10^{17} ionizations, that is, 1 followed by 17 zeros, or the grand total of one hundred million billion ionizations! And that is when only the very slightest proportion of all the atoms in the body are affected. The amount of radiation that each of us receives from our natural surroundings is about 0.1*r* a year. About a quarter of this amount comes from cosmic rays, somewhat less than this from radioactive elements in our bodies, principally potassium, and about half from the soil and rocks.

Other sources of radioactivity that we pick up from our environment are such things as illuminated dials on wrist watches, clocks, automobile dashboards, medical and dental X rays, fluoroscopes, and similar ionizing sources of artificially created radiation. Thus far, the damage done to the hereditary mechanisms of thousands of persons by the misuse of medical and dental X rays has probably been considerably greater than that done by fallout from nuclear explosions.

But ordinary articles which we take for granted are potentially even more risky. It has, for example, recently been shown by Haybrittle in England that the gamma radiation given off by the luminous dials of many ordinary wrist watches is only second to that which diagnostic radiology may contribute to the gonads. Haybrittle found that one modern self-winding wrist watch contained on its dial 2.2 microcuries† of radium. This meant that if

* Named after Wilhelm Konrad Roentgen, the German physicist who discovered the X ray in 1895, for which discovery he was awarded the Nobel prize in 1901.

† A *curie* is the amount of material in which 3.7×10^{10} atoms disintegrate per second, that is to say the transformation of 37,000,000,000 atoms occurring each second in 1 gram ($\frac{1}{28}$ ounce) of radium. A *microcurie* is a millionth of a curie.

the wearer put the watch on his wrist at 8 A.M. and took it off before retiring at midnight, he would receive during those sixteen hours a total of 0.9r per week! This is nearly two thirds of the present maximum permissible exposure to limited parts of the body, such as hands and forearms. A simple calculation will show that in the course of a lifetime an individual would accumulate a considerable amount of radiation from this source alone! The radiation dose to the gonads would be increased by more than 10 percent above that received from background radiation.

These observations have been confirmed in the United States by Chase and Osol who calculate that an illuminated wrist watch worn twenty-four hours a day can deliver to the gonads 1.1r units a year, if the watch is worn facing the gonads. The potentially harmful magnitude of the radiation from the most active watches corresponding to 5r in about five years may be judged in the light of the recommendation of the International Commission on Radiation Protection that no one should receive a dose in excess of 5r by the age of thirty. "When," these investigators write, "one further considers that this radiation is several times greater than natural background radiation and exceeds by more than 100 times that presently received from radioactive fallout, the potential hazard to the wearer of a luminous-dial wrist watch raises the question whether the small benefit that may be received from such a watch is worth the hazard."

It has already been stated that however slight the radiation, it is capable of causing mutations. There is no minimum amount of radiation that must be exceeded before harmful mutations can be induced. It has also been pointed out that more than 99 percent of mutations are harmful. Since mutations constitute the raw material of evolution, without which there could be no evolution, how, then, the reader may well ask, does it come about that good effects can result from mutations?

The answer is that it is not mutations of themselves that produce evolution, but rather the process whereby *favorable* mutations, when they do occur, by increasing the adaptive fitness of the organism to meet the requirements of the environment, enable it better to survive and leave a larger progeny behind it than those who do not possess similar favorable mutant genes. This is the process known as *natural selection*.

Man's mutation rate throughout his history has been quite

adequate to ensure his favorable continuing evolution without the necessity of any artificial speeding up of that rate. In fact, the great danger from artificially produced radiation lies in this, that by increasing the mutation rate, unfavorable mutations would accumulate in such high frequencies that they would endanger the very survival of the species. The compounding hazard being that owing to the extraordinary mastery of the environment that man has achieved, in medical care and in everyday living, he tends to prevent the elimination of unfavorable mutations, to interfere with *selection pressure*, and by this means, in the event of any sizable increase in the numbers of harmful mutations he would contribute further to his own downfall.

It is desirable to be quite clear as to the nature of those mutations. The idea that they would result in numerous monstrosities or freaks is quite erroneous. Undoubtedly some mutations would result in abnormalities of embryonic development, and some developmentally abnormal infants would be born. But these would be the minority. Most mutations are responsible for only slightly detrimental effects.

Paul Dehn, the author of the following parody is, therefore, not altogether sound in his contemporary version of an old hymn:*

Hark, the herald angels sing
Glory to the new-born thing
Which, because of radiation,
Will be cared for by the nation.

However, there are some mutations which in their effects will vary all the way from producing death to sterility or some other serious major defect. Such effects will generally be observable within the first generation. The slight detrimental effects would not be so readily observed, for they will remain concealed in the genetic constitution of successive generations. In some individuals the genetic damage would express itself immediately, even if the mutant gene were carried in the heterozygous state. Such heterozygous damage, as it may be called, is brought about as a result of the fact that detrimental recessives are not usually completely dominated by the normal gene, for some effect—however slight—is usually produced by the abnormal recessive. The effect is ordinarily much smaller than that which the gene exerts in the

* *New Statesman*, August 22, 1958, p. 215.

homozygous state, and yet as a consequence there may be an appreciable decrease in resistance to disease, in length of life, and in fertility in these heterozygous carriers. Every carrier of the mutant gene, and every single descendant who receives it, is in danger of such heterozygous damage. Therefore the number of individuals affected is very great. However weakly expressed, the damage would persist and some of it would be exhibited in each generation in the form either of premature death, the failure to produce the normal number of offspring, or lowered resistance.

The genetic damage done is roughly proportional to the mutation rate. The mutation rate is proportional to the radiation dosage received. If we increase the radiation by 5 percent, then the mutations caused by radiation will also be increased by 5 percent. If, for example, we then add an increase of 7 percent of radiation to the previous 5 percent, we shall have a total dose of 12 percent radiation and a total increase of mutation by another 7 percent to 12 percent. The genetic damage done is cumulative, whether spread out over a short or a long period of time. Large doses of radiation produce more mutations but not more substantially harmful ones.

Clearly, then, if all radiation is capable of producing mutations in the germ cells, there can be no such thing as a safe mutation rate since almost all mutations are genetically damaging. The important consideration is the genetic damage done as a result of the total accumulated radiation dose to the reproductive cells of the individual from his own conception to the time his child is conceived.

In the United States the average age of both parents at the birth of *all* their children is twenty-eight years. Thus, an individual is exposed for approximately his first thirty years (a whole generation) to radiation up to and including the time of the conception and birth of all his children. This is his *total reproductive-life radiation dose*.

The total accumulated thirty-year dose that each individual, on the average, receives has been estimated to be as follows:

<i>Background Radiation:</i> Cosmic rays, naturally occurring	
radioactive substances, etc.	4.3r*
<i>Medical X rays:</i>	3.0r
<i>Fallout:</i> From weapons tests	0.1r
	7.4r

*At high altitudes this would amount to about 5.5r, owing to greater exposure to cosmic rays.

These estimates do not take into consideration such extra sources of radiation as radioactive dials on watches, dashboards, and other instruments, isotopic tracers used in research, Carbon 14 estimation methods, high-power radio and television stations, and the like, not to mention the recently suggested probable mutation effects of increased temperature of the testes in males as a result of wearing trousers!

In 1957 the Swedish workers Ehrenberg, von Ehrenstein, and Hedgran reported the results of an investigation on the scrotal temperature of nude and clothed men. They found the scrotal temperature to be higher by 3.3° C., on the average, in the clothed men as compared with the nude men. They calculate that such an increase in scrotal temperature would increase the mutation rate by about 85 percent! They write, "The fact that our modes of dress have been predominant for several centuries might explain almost half the present load of spontaneous mutations. We thus see how modes of dress based chiefly on sexual taboos might imply genetical hazards 100 to 1,000 times greater than those estimated from different sources of radiation. If the eugenists regard this increase as dangerous, the design of male clothing will have to be reformed, for example, in the direction of the Scottish kilt or of trousers fitted with a codpiece as used in medieval Europe. Garments that tend to press the scrotum against the abdominal skin will be specially hazardous. Further consideration will have to be given to such factors as central-heated rooms and frequent hot baths."

In short, the wearing of trousers, not to mention frequent hot baths and the central overheating to which Americans voluntarily subject themselves for about six months of each year, is probably more dangerous to future generations than the fallout from atomic and hydrogen bombs. The result of all this may be that men will soon be wearing skirts!

If we proceed at the present rate with nuclear tests and medical radiology (and excluding luminous wrist watches) the total accumulated thirty-year dose we are now receiving, on the average, is 7.4r, or 3.1r above the amount of radiation we would normally receive from natural sources. This means that the mutation rate has increased proportionately in countries like the United States in which there is considerable exposure to medical X rays. Among uncivilized peoples the exposure is limited to background radiation and fallout, and except near the islands in the Pacific where

weapons tests have been conducted, their total additional radiation dosage amounts to no more than 0.1 percent of the total, which is less than 2 percent of the total radiation which man is now receiving. It is likely, therefore, that there will be different mutation rates among different populations of man. The reason for this difference underscores the fact that by far the largest dose of radiation, in addition to natural background radiation, is received by civilized man from medical X rays to which he is exposed for diagnostic and therapeutic purposes.

An amount of less than 2 percent of radiation derived from fallout may seem very little. But in fact when that amount is distributed over a world population of 2,750,000,000, it adds up to a very tangible amount of genetic damage. Dr. James F. Crow, geneticist of the University of Wisconsin, has estimated that an exposure of the world's population to 0.1r would result in at least 8,000 children in the first generation born with gross physical or mental defects, or a total of 80,000 in the future generations. In the next generation there would be about 40,000 embryo and infant deaths, or a total of 700,000 in the long run. Dr. Crow thinks that these figures are probably an underestimate.

"A small fraction" of the population turns out to be a very large number of human beings, and incalculably harmful in terms of sheer human tragedy and suffering.

What man's natural mutation rate may be is unknown, but Dr. W. L. Russell of the Oak Ridge Laboratory has shown in the house mouse that 1r produces about 1 mutation in 4,000,000 genes. Comparing this rate with the spontaneous mutation rate which we know for certain genes, such as the hemophilia gene in man, it is estimated that a dose of some 30r to 60r would double man's natural mutation rate.

It should be emphasized that we are not in this chapter concerned with the effects of radiation upon the body, such as burns, cancer, or leukemia, but with its effects upon heredity.

Having briefly considered the effects of fallout upon heredity, let us now proceed to consider the general effects of possible increased radiation upon the heredity of mankind. This can, perhaps, be best illustrated in the following manner:

It is estimated that between 4 and 5 percent of all live children born in the United States suffer from such defects as mental deficiency, epilepsy, congenital malformations, neuromuscular disorders, disorders of the blood and of the glandular systems, skin

TABLE XXXI
ESTIMATES OF SPONTANEOUS MUTATION RATES OF SOME HUMAN GENES

Trait	Mode of Inheritance	Mutation Frequency of Causal Gene (per million per generation)	Region
Achondroplasia	Dominant	45	Denmark
Aniridia	"	5	"
Epiloia	"	8	England
Microphthalmos	"	5	Sweden
Partial albinism (with deafness)	"	4	Holland
Retinoblastoma	"	4	Germany
"	"	15	England
"	"	23	United States
Hemophilia	Sex-linked	20	England
"	"	27	Switzerland & Denmark
"	"	32	Denmark
Muscular dystrophy (Duchenne type)	"	43	England
"	"	45	N. Ireland
"	"	95	United States
Albinism	Recessive	28	Japan
Amyotonia congenita	"	20	Sweden
Color blindness (total)	"	28	Japan
Ichthyosis congenita	"	11	"
Infantile amaurotic idiocy	"	11	"
True microcephaly	"	49	"
Phenylketonuria	"	25	—

Adapted from Penrose, L. S., "The Spontaneous Mutation Rate in Man."

and skeletal disorders, or defects of the gastrointestinal or genitourinary systems. About half of these defects, that is, about 2 per cent—a total of 2,000,000—of the total live births, are of genetic origin and appear before sexual maturity.

While we know something about mutation rates for certain disorders in various populations (see Table XXXI), it is quite another matter for the individual. In every individual, during his lifetime, a certain number of mutations occur due to natural causes. We do not know what the number is. One to four is a fair

guess, with the total number of mutant genes carried in the recessive state by each individual being eight. But whatever the number is, what would be the radiation dose that would produce an equal number of additional mutations, and thus double the total number? It has been estimated that the doubling dose would be somewhere between 30r and 80r. Supposing, now, that the population of the United States were to receive a doubling dose of radiation, what would be the genetic effect upon the population, in relation to the genetic defects mentioned above?

The 2,000,000 genetic defectives would eventually be doubled, provided the doubling dose continued in each generation and the population remained stationary. In the first generation there would be an increase of about 200,000 genetic defectives. These would be the individuals with tangible defects responsible for real personal and social distress. But in addition the concealed genetic damage would be considerable.

If the radiation dosage received by the gonads were 10r for the same population, then there would be about 5,000,000 mutants in the 100,000,000 children born in the next generation.

The Mechanism of Genetic Damage

In addition to producing changes in the structure of genes, that is, mutations, ionizing radiations produce breaks in chromosomes (see fig. 45). The chromosome breaks, like mutations, are caused by the ionizing radiations in the vicinity of the chromosomes or within the chromosomes themselves. The broken surfaces of the chromosomes remain sticky for some time after the breaks have been produced, and any one of the following four things finally ensues:

1. The broken surfaces unite and healing takes place. At the position where the break originally occurred, one or more genes may be damaged or altered and this may give rise to one or more mutations. There may, however, be no damage at the break, in which case no permanent harm has been done.

2. The broken surfaces fail to unite, and the nucleus of the cell now contains several separated fragments of a chromosome. Such broken fragments can replicate themselves, but as soon as the cell starts to divide and passes through one or two cell divisions the fragmented chromosomes tend to break down and the cells to which they have been distributed likewise break down and die for want of the neces-

sary genes. Dividing cells are very much more sensitive to radiation than nondividing or resting cells.

3. The several broken portions of the *same* chromosome may get shuffled around and their broken ends stick together in new ways, or one or more of the fragments may be lost. This is known as *chromosome rearrangement*. The consequences of this may be extremely varied, from death to the organism, or every appearance of mutation, or overtly scarcely noticeable.

4. The several broken portions of any two chromosomes, whether paired or not, may accidentally exchange pieces, and the pieces may be of very different lengths. This abnormal process is known as *translocation*. Translocations tend to result in a high proportion of genetic deaths. Since at the maturation division the abnormal translocated chromosomes are unable to pair with the normal chromosomes, many of the gametes will possess a subnormal number of chromosomes and genes. Translocations result either in death or reduced fertility of those who inherit them, and in either case are inherited like dominant genes.

What happens as a result of chromosome rearrangements and translocation is that the sequences of the purine and pyrimidine bases, adenine, guanine, cytosine, and thymine, between the helices that comprise the DNA or deoxyribonucleic acid molecule (see pages 33–39) get disarranged. Since the genetic information that is transmitted by the hereditary mechanism lies coded according to the sequence of these bases, any change in their order or in their structure will be reflected in changes in the information transmitted, and as we have abundantly emphasized, such changes, whether they be in the form of mutations or fragmented chromosome pieces which have attached themselves to pieces from the same or different chromosomes, are usually deleterious in their effects. These changes will occur not only in the reproductive cells but also in the somatic or body cells, and of course will cause damage in the body cells which may result in all sorts of more or less serious disorders. In many cases they will have the effect of increasing the frequency of disorders and diseases, and deaths from these.

Chemical defects in the genes, whether in the reproductive or the body cells, result in defective reactions or in the blocking of necessary chemical reactions, so that there tends to be either defective development or a failure to develop altogether as a consequence of the blocking process, which is itself simply a failure in the development of a necessary chemical substance. Such a failure, for example, is believed to be responsible for albinism. In the

formation of the pigment melanin, for example, a series of complex chemical steps must occur in an orderly manner. These steps are produced by a series of enzymes. In the case of albinism one of these enzymes fails to develop, and the series of steps which normally leads to melanin formation is blocked and the resulting individual fails to develop any pigment at all. The enzyme thought to be involved is one that acts either on 3,4-dihydroxyphenylalanine or possibly on tyrosine. At its most elementary the disturbance is essentially one of the intake, consumption, and expenditure of energy, that is, metabolism: the energy relations of the gene are disturbed.

Some Conclusions

The United Nations Scientific Committee on Effects of Atomic Radiation, in its report published in August, 1958, stated:

The knowledge that man's actions can impair his genetic inheritance, and the cumulative effect of ionizing radiation in causing such impairment, clearly emphasize the responsibilities of the present generation, particularly in view of the social consequences laid on human populations by unfavorable genes.

Besides increasing the incidence of easily discernible disorders, many of them serious but each comparatively rare, increased mutation may affect certain universal and important "biometrical" characters such as intelligence or life-span.

As these words were being written two U.S.S.R. scientists, M. N. Livanov and D. A. Biryukov reported to the United Nations Second International Conference on Peaceful Uses of Atomic Energy held at Geneva in September, 1958, that profound effects are produced upon the brain and central nervous system by ionizing radiations. Since children are more sensitive to ionizing radiations than adults, the functioning of the nervous system and intelligence would be affected in a large proportion of the young, so that those that had not been hereditarily affected might be brought down to the level of those who had. But, of course, a large number of adults at every age would also be detrimentally affected by ionizing radiations affecting the nervous system and intelligence.

Insofar as the hereditary changes deleteriously affecting intelligence and the functioning of the nervous system are concerned, these unfortunately are likely to be of the kind that are not detectable in the form of easily seen gene effects. They could, there-

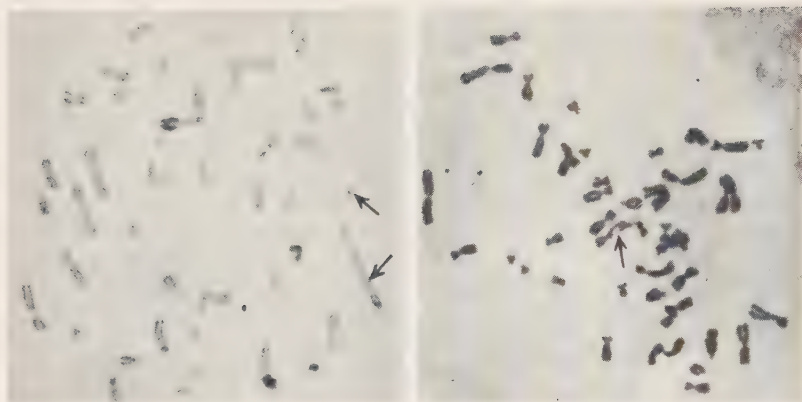


FIG. 45. X-Ray Induced Chromosome Breakage in Man. Arrows indicate places where breakage of chromatids is evident. Exposure 50r. There were 21.6 percent of cells with such chromosome breaks induced by the X radiation. (Courtesy Dr. Michael A. Bender, Oak Ridge National Laboratory.)

fore, only be picked up in the form of significant increases in the incidence of such disorders. Exposure to continued small doses of radiation will produce correspondingly small effects in most of the members of the population and large ones in a comparatively small number of individuals. This kind of hidden damage is much more difficult to isolate and deal with. Hence, much more research is necessary, particularly of the kind that will lead us to detect this kind of hidden genetic damage. The danger of rendering ourselves too stupid, as a result of exposure to ionizing radiations, to arrive at such a method is not for the immediate future very great.

Meanwhile each of us can take certain simple practical steps to reduce the amount of radiation to which we and our children are exposed.

We can begin with all timepieces, clocks, and watches that are radioactive, that is, that have their dials painted with a radium paint—and this includes all radioactive dials on the dashboards of automobiles; they should be jettisoned.

We should see to it that legislation is passed forbidding anyone to use any apparatus producing ionizing radiations who is not properly qualified to use such apparatus.

No X-ray apparatus should be manufactured by companies unlicensed to do so. Licenses to manufacture X-ray apparatus should

be issued only to those manufacturers who meet the proper specifications for the manufacture of such installations.

A person shall not be considered qualified to use X-ray apparatus for diagnostic or therapeutic purposes, no matter what other diplomas he may have, unless he has been specially certified to do so after having undergone the proper training and taken the proper examinations. This would at the present time exclude most medical men and most dentists.

All medical and dental X-ray installations should be subject to a quarterly checking in order to control the dosages, filtration, and the safety in general of those who are exposed to its radiations.

In a report on *The Biological Effects of Atomic Radiation*, published by the National Academy of Sciences and the National Research Council, the following recommendations are made:

1. That, in view of the fact that total accumulated dose is the genetically important figure, steps be taken to institute a national system of radiation exposure record-keeping, under which there would be maintained for every individual a complete history of his total record of exposure to X rays, and to all other gamma radiation. This will impose minor burdens on all individuals of our society, but it will, as a compensation, be a real protection to them. We are conscious of the fact that this recommendation will not be simple to put into effect.

2. That the medical authorities of this country initiate a vigorous movement to reduce the radiation exposure from X rays to the lowest limit consistent with medical necessity; and in particular that they take steps to assure that proper safeguards always be taken to minimize the radiation dose to the reproductive cells.

3. That for the present it be accepted as a uniform national standard that X-ray installations (medical and nonmedical), power installations, disposal of radioactive wastes, experimental installations, testing of weapons, and all other humanly controllable sources of radiations be so restricted that members of our general population shall not receive from such sources an average of more than 10 roentgens, in addition to background, of ionizing radiation as a total accumulated dose in the reproductive cells from conception to age 30.

4. The previous recommendation should be reconsidered periodically with the view to keeping the reproductive cell dose at the lowest practicable level. If it is feasible to reduce medical exposures, industrial exposures, or both, then the total should be reduced accordingly.

5. That individual persons not receive more than a total accumulated dose to the reproductive cells of 50 roentgens up to age 30 years (by which age, on the average, over half of the children will have been

born), and not more than 50 roentgens additional up to age 40 (by which time about nine tenths of their children will have been born).

6. That every effort be made to assign to tasks involving higher radiation exposures individuals who, for age or other reasons, are unlikely thereafter to have additional offspring. Again it is recognized that such a procedure will introduce complications and difficulties, but this committee is convinced that society should begin to modify its procedures to meet inevitable new conditions.

The Committee underscores its conclusion that the public be protected, by whatever controls necessary, from receiving a total reproductive-lifetime dose (conception to the age of thirty) of more than 10r of artificially induced radiation to the reproductive cells.

The public should see to it that it secures such protection.

As Linus Pauling has said, "We are the custodians of the human race. We have the duty of protecting the pool of human germ plasm against willful damage." We must, therefore, do everything necessary to see to it that all atom bomb testing, which releases so many menacing radioactive substances, will be immediately and forever discontinued.

Among these menacing radioactive substances is carbon 14. Carbon 14 has a life of 8,070 years, and is damaging to human beings throughout that time. It has been estimated by Linus Pauling that the carbon 14 thus far released by the bomb tests will in the long run produce about one million seriously defective children, about two million embryonic and newborn deaths, and will produce minor hereditary defects in many millions of individuals.

These are some of the facts about the effects of radiation upon heredity. For the rest the question is whether you, the reader, are going to remain part of the problem which they present or whether you are going to make yourself a part of the solution.

APPENDIX A

Inherited Disorders of Man

THE CLASSIFIED list of inherited disorders which is presented in the following pages is designed to give the reader some idea of the kinds of disorders that are largely determined by genetic factors, and also to provide him with a short statement of the mode of inheritance involved. While fuller than any similar list hitherto published, it is to be understood that the present list is by no means a complete census of all known hereditary conditions—not to mention those whose mode of inheritance is unknown. The list is, however, fairly representative.

A word of caution: In consulting this list it should be remembered that many of the conditions listed are sometimes produced in ways which have little or no connection with genetic factors. Intrauterine environmental conditions and extrauterine environmental conditions of many varieties may produce disorders which in every way resemble those which are principally due to defective genes. Such copies of the genetically induced condition are known as *phenocopies*—and should be carefully distinguished from the genotypically produced disorders. Furthermore, some disorders arise quite sporadically for reasons which are often obscure, and these, too, should be distinguished from the hereditary conditions.

The classification is based fundamentally on the organs or organ systems affected. Thus, if the reader knows what organ or organ system is involved in the condition he wishes to look up, all he has to do is to turn to the appropriate heading. If, however, all that he knows is simply the name of the disorder he may save time by looking into the index which will tell him on what page the condition is listed. In the index he will find the condition listed separately under its ordinary and its technical names. However, it has not been possible to classify all the conditions listed under organs or organ systems—for example, allergies or disorders of metabolism—but this should make no great difficulty for the reader.

Wherever possible, the incidence of the condition in the population in general has been given, as well as the sex differences in incidence. Where the former is not given, the evidence is not quite clear; where the latter is

not given, it may, in general, be assumed that the condition appears with equal frequency in both sexes. Where complete sex-linkage obtains, it is to be understood that the condition appears exclusively in males.

Alternative modes of inheritance can sometimes occur in addition to those given.

NOTE: In this list "dominant" will be abbreviated as *D* and "recessive" as *R*.

ALLERGIES

Angioneurotic edema (Sudden swellings, which may lead to death when occurring in throat)

D

Asthma

Probably D, with about 40 percent penetrance

Risk: With 1 parent asthmatic, 50 percent of their children will be carriers, and of these, 13 percent will exhibit asthma and vasomotor rhinitis and 7 percent hay fever or atopic dermatitis (Bessner's prurigo).

Urticaria (Hives)

Possibly irregular R

BLOOD AND VASCULAR SYSTEM

ABO hemolytic disease (ABO incompatibility between mother and child; where, for example, mother is blood group O and the child is A or B, the cells of the fetus tend to be agglutinated by anti-A or anti-B antibodies in the mother's serum. The condition is usually not serious, though it is 3 times more common than Rh hemolytic disease.)

Incidence: 1 in 71 births, and may arise in 7 percent of AB incompatible mother-child pairs

Risk: Thought to be responsible for the loss of about 25 percent of A children expected from marriage of A × O

Acanthocytosis (Malformed, crenated red blood cells, may be accompanied

by atypical retinitis pigmentosa and evidences of involvement of nervous system)

Probably R

Acatalasia (Oral gangrene, absence of catalase in blood)

Probably R

Afibrinogenemia (Congenital fibrinogen deficiency—blood-plasma globulin—resulting in severe hemorrhagic symptoms)

Probably R

Preponderance of males

In some individuals carrying the gene in heterozygous state the gene may express itself in a mild form of the disorder in a relative deficiency of fibrinogen (fibrinopenia) rather than in complete absence of fibrinogen.

Agammaglobulinemia (Extreme reduction in immunity-conferring gamma globulin)

(i) Infant type

(ii) Adult type

Sex-linked

Alder's anomaly (Coarse granulation of white blood cells)

Probably D

Arteriosclerosis

There is a strong tendency in some families for many of its members to develop this disorder, indicating a genetic basis, which is at present not clear.

Christmas disease (Resembling mild to moderately severe hemophilia)

Sex-linked R

Combined hemophilia and Christmas disease

Sex-linked

Combined hemophilia and parahemophilia

Sex-linked R *plus* autosomal R
Largely limited to males

Coronary xanthomatosis (Yellow deposits of nodules in the heart valves and/or the coronary arteries resulting in angina pectoris)

D

Endocardial fibroelastosis (Breakdown of inner lining of heart; death within first decade)

Possibly mutant D

Familial eosinophilia (Increase in circulating polymorphonuclear eosinophils, or readily staining leucocytes or white cells)

Probably D

Hageman trait (Nonhemorrhagic disorder of coagulation of blood with prolonged clotting time of venous blood)

Probably R

Heart Disease, congenital

(i) *Patent ductus arteriosus* (Failure to close of the fetal artery leading from pulmonary to aortic artery). Probably D

(ii) *Auricular septal defect*. Probably D

(iii) *Lutembacher's syndrome* (Mitral stenosis and interauricular defect). Probably R

(iv) *Tetralogy of Fallot* (Pulmonary stenosis or atresia, interventricular defect, and hypertrophy of right ventricle). Probably R

Risk: Of any variety of cardiac malformation in children 1 in 50; likelihood of recurrence of identical malformation 1 in 100

Hemolytic disease of the newborn (*Erythroblastosis fetalis*) (Due to incompatibility of the mother's genotype Rh-negative and the baby's genotype Rh-positive)

Incidence: 1 in 200 pregnancies

Risk: If father carries an Rh-positive gene and an Rh-negative gene, there is an even chance that sibling of affected child will be normal. If father's genes are both Rh-positive, chances of normal future pregnancies are poor.

First child usually escapes

Hemophilia (Excessive and prolonged hemorrhage following trivial injury or occurring spontaneously due to deficiency of thromboplastinogen—antihemophilic globulin)

Sex-linked R

Incidence: 1 in 25,000

Mutation rate: 1 in 50,000 in female, 1 in 10,000 in male

Hemorrhagic disease, autosomal (Prolonged hemorrhage following injury, with deficiency of antihemophilic globulin)

Unlike hemophilia this condition is *not* sex-linked, but upon the basis of the few cases recorded seems to be sex-limited occurring only in females.

Homozygous hemoglobin C disease (Hemolytic anemia, enlarged liver and spleen, etc.)

Probably irregular R

Largely limited to Negroes

Hyperchromic anemia (*Fanconi syndrome*) (Blood affected in same way as in pernicious anemia but there are accompanying skeletal defects, microcephaly, strabismus, brown pigmentation of skin, undeveloped gonads; usually fatal in childhood)

Probably R

Preponderance of males

Hypertension, essential (High blood pressure)

Probably irregular D expressing itself only under appropriate environmental conditions

Hypochromic, microcytic anemia (Pallid small red blood cells, often with enlargement of spleen and liver; frequently terminates fatally at early age)

- Probably sex-linked, but may also be autosomal D with sex-limited effect
- Hypoplastic anemia of childhood* (Great reduction in blood cells, terminating in death)
Probably R
Aplastic form: Even more extensive reduction in leucocytes and platelets. Probably R
Incidence: Extremely rare
- Hypotension* (Low blood pressure)
Probably D
- Jaundice, congenital hemolytic (spherocytic anemia, chronic acholuric jaundice)* (Sphere-shaped red blood cells with marked rate of blood destruction)
D
Incidence: 1 in 15,000
- Leukemia* (Abnormal increase in leucocytes, or white blood cells)
Genetic basis unclear, but often associated with abnormal number of chromosomes
- Neutropenia* (Chronic decrease in number of polymorphonuclear neutrophils, or white cells)
D
- Ovalocytosis (elliptocytosis)* (Oval or elliptical erythrocytes—red blood cells—in blood)
D
Incidence: 1 in 3,000
- Parahemophilia* (Deficiency of plasma labile Factor V—proaccelerin—necessary for conversion of prothrombin to thrombin; resembles hemophilia clinically)
R. In the heterozygous state the effects of the single gene can be picked up in the carriers since they have a significant reduction of Factor V.
- Pelger's nuclear anomaly* (Nonsegmentation of the normally segmented nuclei of polymorphonuclear leucocytes)
D
- Pernicious anemia (primary anemia, Addison's anemia)* (Chronic progressive reduction of red blood cells)
A genetic tendency evident in many families, but mechanism unclear.
Incidence: 1–2 in 10,000
- Phlebectasis (varicose veins)*
Irregular D
- Rheumatic heart disease*
R
- Sickle-cell anemia* (So called because when drop of blood from affected individual is deprived of oxygen, red blood cells assume sicklelike shapes)
(i) *Sickle-cell disease* (Severe anemia, usually fatal in childhood). Homozygous D. Incidence: 1 out of 500 American Negroes.
(ii) *Sickle-cell trait* (Mild anemia). Heterozygous D
Largely limited to peoples of Negroid origin
- Stuart clotting defect* (A hemophilia-like condition with moderate bleeding tendency and prolonged plasma clotting time)
Incomplete R
- Telangiectasia, hemorrhagic (Osler's disease)* (Dilatation of terminal portions of small vessels with repeated bleeding)
D, with reduced penetrance; in homozygous state may be lethal
- Thalassemia (Cooley's anemia, Mediterranean anemia)*
(i) *Thalassemia minor* (Mild anemia with increased fragility of red blood cells and increased number of target and oval cells). Heterozygous, incomplete D
(ii) *Thalassemia major* (Progressive fatal hemolytic anemia). Homozygous D
- Thromboangiitis obliterans (Buerger's disease)*
Probably R
- Thrombocytopathic purpura (Glanzmann's thrombasthenia)* (Normal number but abnormal appearance of platelets, normal coagulation time, poor clot retraction)
Probably D

Thrombocytopenic purpura (Werlhof's disease) (Bleeding from and into the skin in small spots and patches, great reduction in blood platelets, poor clotting)

D

BONES AND JOINTS

Achondroplasia (chondrodystrophy, chondrodystrophia fetalis) (Stunting of skeletal growth owing to anomalous cartilaginous growth)

D; some cases possibly R

Incidence: 1 in 15,000

Acro-osteolysis (Shrinkage of the bones, usually of the extremities)

Irregular D

Onset: Any time from birth

Amputations, congenital (Absence of arms and/or legs)

R, incomplete D, and D genotypes may exist.

Arthro-onychodysplasia (Congenital dystrophy of nails, elbows, and absence of kneecap)

D, of variable expressivity

Chondroectodermal dysplasia (Ellis-Van Crevald syndrome) (Disturbances in development of hair, teeth, skin, and other ectodermal derivatives; polydactyly; achondroplasia)

Probably R

Clubfoot, congenital

Irregular D, probably sex-influenced, and exhibiting low penetrance and great variability in expressivity

Twice as frequent in males as in females.

Incidence: 1 in 1,000

Risk: For siblings of consanguineous relationship, 25 percent; for sibling with one affected parent, 10 percent; for sibling with unaffected parents, 3 percent

Deep acetabulum and intrapelvic protrusion

Probably D

Diaphyseal aclasis (Multiple cartilaginous exostoses, or bony growths)

D

Of those affected, 70 percent are males.

Dysplasia epiphysitis punctata (chondrodystrophia calcificans congenital)

Possibly irregular D

Slightly more frequent in females

Enchondromas (Ollier's dyschondroplasia) (Multiple deformities of tubular bones)

Possibly D, with very low penetrance

Hallux rigidus (Stiff big toe)

Probably D

Hallux valgus (Bunion)

D

Hammer toe (The second toe is usually involved)

D

Hip, congenital dislocation of

Probably D, with lack of penetrance due to sex and environmental factors; intra- and extra-uterine environmental factors involved, for example, twice as many cases occur in winter as in summer, and maternal age is a significant factor.

85 percent of the affected infants are females.

Parietal foramina, enlarged (Defects in the posterior angles of the parietal skull bones)

D

Osteitis deformans (Paget's disease) (Thickening and rarefaction of the bones)

Probably irregular D, may be sex-linked in some cases

Slightly greater incidence in males

Osteochondritis deformans juvenilis of the hip (Legg-Perthes' disease) (Disease of the hip joint appearing in children between ages of 8 and 12)

D, sometimes R

Incidence: much higher in boys than in girls

Osteochondrodystrophia deformans (Morquio's disease) (Deformation of skeleton with dwarfing)

Usually R, sometimes sex-linked, and occasionally D

Osteogenesis imperfecta (van der Hoeve syndrome, Lobstein's disease, fragilitas ossium) Extreme fragility with multiple fractures of bones, blue sclerae of eyes)

(i) *Congenital*. Prognosis very poor

(ii) *Osteogenesis imperfecta tarda*.

Divided into two types: I, in which fractures begin at birth, and II, in which fractures begin between 2 and 3 years. In I they continue throughout life; in II they begin to decline in frequency at about puberty. Otosclerosis develops in 25 percent of cases.

D, with pleiotropic effect

Incidence: 1 in 50,000

Osteopetrosis (Albers-Schönberg disease, marble bones) (Hardening followed by softening and great fragility of bones)

(i) *Malignant form*. Probably R

(ii) *Benign form*. D, with reduced penetrance

Osteopoikilosis (spotted bones) (Islands of compact bone developed in the spongy, or cancellous, portions of bones; symptomless)

D, possibly R in some cases

Pes planus (Convex valgus foot—flat-foot)

D

Radial reduction (Partial or complete absence of radius and aplasia of radial rays of hands and fingers)

D

A very minor defect in one individual may be followed by a much more severe form in later generations.

Radiohumeral, ulna-humeral synostosis, congenital (Fusion between the humerus and the ulna or radius)

D, irregular D, R sex-linked or male sex-limited

Over 50 percent of cases are bilateral. If not complete, a rudimentary elbow joint may be formed.

More frequent in males

Radio-ulnar defect (Absence of radius and ulna)

R or sometimes possibly irregular D

Incidence: 1 in 75,000

Rickets, Vitamin D resistant (Familial hypophosphatemia)

Sex-linked D with complete penetrance. Possibly cases occur due to autosomal D or R.

Scapulae, elevated (Sprengel's deformity) (High position of the shoulder blades owing to their failure to descend in development)

D and R genotypes have been described.

Spina bifida (Cleft spinal column)

(i) *Spina bifida aperta*. Preponderant in males. Incidence: 1 in 400

(ii) *Spina bifida occulta*.

The hereditary mechanism is unknown; it would appear that many genotypes for the condition may exist.

Risk: For siblings, of having spina bifida, anencephaly, or hydrocephaly, 4 percent.

Split-foot

Irregular D. Appears to be the effect of the same gene as for cleft-hand. Close association exists between syndactyly and cleft-foot.

Spondylitis, ankylosing (Hardening of the joints of the spinal column and sacroiliac region with rigidity of spine)

D, with 70 percent penetrance in males and 10 percent penetrance in females. When affected parent is mother, penetrance is almost total in offspring of both sexes.

Tibial defect (Defective tibia)

D

Ulnar defects (Total or partial absence of ulna with defects of ulnar rays)

Sporadic

EARS AND HEARING

Atresia of auditory meatus (Developmental failure of earhole to open, usually with rudimentary auditory ossicles)

D

Auricular appendages (Congenital tumors on external ear, usually non-malignant)

Probably D

Auricular fistula (Congenital hole, usually ending in a shallow blind depression where front of rolled part of ear meets skin of face)

Usually irregular D

Cat's ear (Diminished size of ear, projecting sideways)

D

Deaf-mutism

This condition is dependent upon the way two pairs of genes, *D* and *d*, *E* and *e*, interact in such a way that *D-E* produce normal hearing, while any other combination produces children who are born deaf. This is an example of *duplicate recessive epistasis*. When a gene of one pair masks the expression of the genes of another pair it is said to be *epistatic* to the other pair.

50 percent of deaf-mutism is due to uterine environmental causes.

Incidence: 1 in 3,000

Hypertrichosis auricularum (Overgrowth of hair on external ear)

Almost certainly a D sex-limited trait, formerly thought to be transmitted by the Y chromosome

Confined to males

Labyrinthine deafness (Inner-ear deafness)

D

Malformed ears associated with malformation of genitourinary tract (Ears show variety of defects and one kidney usually defective, but any part of the genitourinary system may be affected.

D with about 70 percent penetrance

Microtia (Small ears)

Probably irregular D or possibly sex-linked

Otitis media (Middle-ear inflammation)

Undoubtedly has a genetic basis in many cases, but mode of inheritance not understood.

Otosclerosis (Development of spongy bone in labyrinthine capsule, eventually leading to middle-ear deafness)

D, with reduced penetrance

More frequent in females

Incidence: 1 in 500

Onset: After puberty, usually in adult life

EYES

Albinism, ocular. See *Albinism*, under *Skin*.

Amaurosis congenita, Leber (Total or almost total blindness, involving degeneration of retina and sometimes optic nerve)

Heredofamilial R

Anophthalmos (Congenital absence of eyes)

Appears to be R; probably occurs in sex-linked R form in association with some forms of mental deficiency

Cataract

(i) *Congenital*. Usually D

(ii) *Postnatal*. Generally D

Whether "senile" cataract is genetically influenced is unknown.

Chorioid defects (Defects of the middle coat of the eye)

(i) *Congenital macular coloboma* (Defective development of choroid). D

(ii) *Chorioideremia* (Absence of choroid). Sex-linked R

(iii) *Chorioidal sclerosis* (Hardening of choroid).

(a) Generalized hardening. Usually D

(b) Central hardening. Usually R

- (iv) *Sarcoma* (Tumor). Has been observed as a D

Color blindness

- Sex-linked R

Cornea defects

- (i) *Microcornea*. D
 (ii) *Cornea plana* (Flattened cornea). D
 (iii) *Cornea keratoconus* (Conical). D
 (iv) *Corneal vortex-veil*. D
 (v) *Corneal opacity, congenital*. R
 (vi) *Macular dystrophy*. R
 (vii) *Granular dystrophy*. D
 (viii) *Latticelike dystrophy*. D

Epicanthus (Fold of skin over inner angle of eye, as in the Mongoloid major group)

D

This is not a disorder, but a physical trait, sometimes associated with a disorder, as in mongolism.

Fundus dystrophy (Degeneration of the back of the interior of the eyeball)

D

Onset: At about age 40

Glaucoma (Pressure of fluids in eyeballs, often leading to blindness)

- (i) *Buphthalmos, congenital or infantile*. R, occasionally D
 (ii) *Minimal glaucoma*. D
 (iii) *Absolute glaucoma*. D, sometimes R

Hemeralopia (Day-blindness) (Inability to see clearly in bright light, usually with color blindness)

R, sometimes partially sex-linked

Iris defects

- (i) *Aniridia* (Absence of iris). D with variable expression. Incidence: 1 in 100,000
 (ii) *Hypoplasia* (Incomplete development). D, sometimes sex-linked R, or R
 (iii) *Flocculi iridis* (Woolly iris). D
 (iv) *Persistent pupillary membrane* ("Pinhole" pupil). D

Lens defects

- (i) *Congenital dislocation*. D
 (ii) *Delayed dislocation*. D

- (iii) *Congenital dislocation with displaced pupil*. R

Macular dystrophy (Degeneration of the macula lutea, or the sensitive yellow spot of the retina)

D and R forms have been recognized, while a Sex-Linked R form associated with color blindness also is recognized.

Microphthalmos (Small eyes)

- (i) *Pure type* (With no associated defects). Probably R
 (ii) *With cataract*. D and R genotypes occur.

Mirror reading

Sometimes D

Myopia (Nearsightedness)

- (i) *Extreme*. R
 (ii) *With night blindness*. R and also sex-linked R

Nyctalopia (Night blindness)

- (i) *Congenital stationary night blindness*. D
 (ii) *With myopia*. Sex-linked. Virtually limited to males
 (iii) *With extreme myopia*. R

Nystagmus (Quivering of the eyes)

Sex-linked, D when accompanied by head twitching

Ocular paralysis (Paralysis of eyeball-moving muscles, thus immobilizing eyes)

D and sex-linked genotypes have been described.

Optic nerve atrophy (Degeneration of optic nerve terminating in blindness)

- (i) *Congenital*. D
 (ii) *Childhood type*. R
 (iii) *Adult type* (*Leber's disease*). Sex-linked R, sometimes D

Presbyopia (Farsightedness)

D

Progressive ophthalmoplegia (Drooping of an eyelid, then restriction of ocular movement)

D

Ptosis (Drooping of eyelids)

D

Retinitis pigmentosa (Chronic inflammation of retina with atrophy, etc.)

Usually R, sometimes D, and occasionally sex-linked

Incidence: 1 in 10,000

Retinoblastoma (*Glioma retinae*) (Tumor of retina)

D, with somewhat reduced penetrance

Incidence: 1 in 33,000

Risk: Only 25 percent of affected parents transmit the condition to their offspring because the mutation in many cases has arisen in the somatic cells and not in the reproductive ones. The chance that a child would inherit the trait is about 40 percent, instead of the 50 percent possible if the dominant gene were characterized by complete expressivity.

Strabismus (Cross-eyes)

R and D genotypes have been described.

Risk: If one child has the condition and the parents are normal, the risk that another child may show the condition is 10 percent; with one parent affected it is 17 percent.

Unpigmented eyes (The eyes appear to be pink owing to the presence of blood vessels, but the eye is unpigmented. The condition is limited to the eyes alone.)

Sex-linked

Waardenberg's syndrome (1, wide separation of eyes with lateral displacement of inferior lacrimal punctum, 2, hyperplastic broad nasal root, 3, hyperplasia of medial portions of eyebrows, 4, partial or total heterochromia iridium, 5, congenital deafness or partial, unilateral deafness, and 6, white forelock)

D

Incidence: 1 in 42,000

FINGERS AND TOES

Apical dystrophy (Absence of terminal portions of second to fifth fingers)

D

Arachnodactyly (Abnormally long fingers)

D, with pleiotropic effect

Brachydactyly (Short fingers)

D

Brachymesophalangy (Short middle fingers and/or toes)

D

Brachymorphy with spherophakia (*Marchesani's disease*) Short limbs and digits with small, distorted lens of eye

Incompletely D, possibly R

Brachytelephalangy of the thumbs (Short thumbs)

D

Camptodactyly (Finger or fingers bent toward palm)

Irregular D

Clinodactyly (Bent fingers toward thumb side)

D

Ectrodactylism (Partial or complete absence of fingers)

D

Hyperphalangy of the thumb (Three instead of the normal two phalangeal bones)

D

Madelung's deformity ("Bayonet" hand, shortened forearm, short stature)

Probably D

Polydactylism (Reduplication of fingers)

Usually D, with variable penetrance and expressivity

Split-hand (Lobster-hand)

Irregular D

Incidence: 1 in 90,000

Symphalangism (Bony or fibrous union of joints of fingers or toes)

D

Syndactyly (Fusion of the fingers)

Usually D

Almost twice as frequent in males as females

GASTROINTESTINAL TRACT

Idiopathic megacolon (*Hirschsprung's disease*) (Enlargement of large, and sometimes portion of small, intestine;

nearly always fatal unless surgical correction, by rectosigmoidectomy, is made)

Genetically determined in all cases, but mechanism not understood

Two thirds of the cases are males

Incidence: 1 in 50,000

Risk: A subsequent brother has a 1-in-5 chance of being affected.

Polypsis of large intestine

(i) *Familial intestinal polyposis* (Polyps in colon and rectum which may be malignant). D

(ii) *Peutz's syndrome* (Polyps, mostly in small intestine, but also in stomach, colon, and rectum. There is spotty pigmentation of mucosa of cheek, skin of face, fingers, and toes). D

Risk: Children of affected individuals have a 1-in-2 chance, in both types of polyposis, of inheriting the condition.

Pyloric stenosis, congenital

There is undoubtedly a genetic influence involved but its mechanism is unclear.

80 percent of the cases are male.

Incidence: 3 in 1,000

GENITOURINARY SYSTEM

Cryptorchidism (Undescended testes)

R

Incidence: 1 in 30 boys under 14 and 1 in 250 men over 21

Cystic kidney

(i) *Congenital*. R. Causes death at or shortly after birth

(ii) *Adult type*. D. Onset: At about age 40

Eunuchoidism (Absence of testicles)

Often familial

Hermaphroditism, pseudo (Where only one type of sex gland tissue occurs, either testicular or ovarian but never both)

(i) *Male*. Sex-linked

(ii) *Female*. Probably R

Intrauterine environmental factors

undoubtedly important in many cases.

7 to 8 times more frequent in male

Incidence: 1 in 1,000

Hermaphroditism, true familial (Where both male and female sex gland tissue occurs)

Sex-linked, sex-influenced, and R forms occur, as well as phenocopies of intrauterine origin. Most of the cases are genetic females.

Incidence: 1 in 1,000

Hypospadias (Opening in under-wall of penile urethra)

Irregular R

Incidence: 1 out of every 1,000 males

Klinefelter's syndrome (Apparent male who is sterile, often with marked breast development, and always with small gonads after puberty)

Usually chromatin positive, and characterized by two X chromosomes and a Y chromosome

Incidence: Probably 1 in 400

Nephrosclerosis and essential hypertension (Hardening of the kidney with increased blood pressure)

D

Renal dysplasia (Abnormal development of kidney)

D

Turner's syndrome (Female with developmental failure of ovaries and complete absence of ova, absence of menstruation, and poor development of secondary sexual characters)

Chromatin negative, but not an instance of sex reversal. Turner's syndrome is due to the presence of only one X chromosome and no other, the total chromosome count being 45.

Uterus, absence of

Irregular R

GLANDULAR DISORDERS

Diabetes, false

(i) *Diabetes insipidus*. Usually D, sex-linkage also recognized

(ii) *Renal glycosuria*. Usually D

Preponderant in males

Diabetes mellitus (Excessive output of sugar in urine, etc.)

R, with variable penetrance

Incidence: 1 in 40

Risk: For child of affected parent, 10 to 15 percent; where parents normal, 5 percent; for sibling, one parent affected, 12.5 percent.

Exophthalmic goiter (*Graves' disease*)

(Overactivity of thyroid gland, tenseness, palpitation, high metabolic rate, protrusion of eyeballs)

R, with partial penetrance and sex-limitation to women

Preponderant in females

Goiter, simple (Nontoxic thyroid deficiency)

R, with penetrance of about 1 percent in homozygous males and about 33 percent in homozygous females, and as in *Graves' disease* in another one third of females, while the remainder are unaffected

Myxedema (Thyroid deficiency in adult with greatly reduced metabolism, etc.)

Genetics unclear

Preponderant in females

HAIR AND NAILS

Baldness

(i) *Alopecia* (Pattern baldness). Sex-limited D

(ii) *Alopecia areata* (Patch baldness). D

Grayness, premature

D

Hirsutism, familial idiopathic (Masculine type of hair growth in females of Mediterranean ancestry)

Sex-limited D

Due to inherited hyperfunction of adrenocortical hormones. Can be relieved with prednisone.

Monilethrix (Brittle, very short, or thin and ragged hair)

Irregular D

Pili torti (Twisted hair)

Possibly irregular D

Generally appears in children who have been previously bald for the first year or two

Nails, defects of

(i) *Partial or complete absence*. D or R

(ii) *Thick nails with angular protrusion*. D

(iii) *Thick nails and skin of palms and soles*. D

(iv) *Spotted nails, bluish-white*. D

(v) *Milky-white nails*. Incomplete D

(vi) *Thin nails*. D, with varying penetrance

Nail-patella syndrome (Poorly formed fingernails with absent or small kneecaps, often linked with one or other of the ABO blood groups)

D

HEAD AND NECK

Acrocephalosyndactyly (*Apert's syndrome, acrobrachycephaly*) (Head flattened from back to front with high vault, broad forehead, extreme width between eyes—hypertelorism—webbing of fingers and toes)

Possibly irregular D or R

Cleidocranial dysostosis (Maldevelopment of collarbones and incomplete hardening of skull bones)

D, with good penetrance and variable expressivity

Craniofacial dysostosis (*Crouzon's disease*) (Prominence of forehead, beak-like nose, projecting jaws, bulbous eyes—exophthalmos—and squint)

Irregular D, with fair penetrance

Dacryocystitis (Inflammation of the tear—lacrimal—sac)

D

Dacryostenosis, congenital (Narrowing or stricture of the nasolacrimal duct)

D

Facial hemiatrophy (Wasting of one side of face)

In some cases probably R

Hydrocephalus (Congenital progressive enlargement of cerebral portion of head owing to excessive accumulation of fluid in ventricles of brain)

R

Risk: Where both parents carry the gene 1 to 4 percent of children can be expected to be hydrocephalic

Incidence: 2 in 1,000

Hypertelorism (Extreme distance between the eyes)

Sometimes D, sometimes R

Klippel-Feil syndrome (Shortened neck, etc.)

D, may be with reduced penetrance.

Mandibulofacial dysostosis (Treacher-Collins syndrome) (Fishlike face, malformed ears, etc.)

Irregular D

Microcephaly (Very small head and narrow forehead, large ears, prominent nose)

Probably R

Oxycephaly (Peaked crown of skull)

Sometimes D, sometimes R

Has been observed in association with hereditary hemolytic jaundice

LUNGS AND CHEST

Bronchiectasis

(i) *Congenital* (Cavities of bronchi and bronchioles)

(ii) *Acquired* (Dilatation of bronchi and bronchioles)

The familial incidence of this condition has long been known, and it would seem that D and R genotypes occur.

Bronchitis (Inflammation of mucous membranes of bronchial tubes)

A tendency to both acute and chronic bronchitis may have a genetic basis.

Emphysema of lungs (dilatation of alveoli of lungs)

Familial incidence has been reported.

Funnel chest (Conical depression of chest due to sinking in of the sternum)
D

Pneumothorax, spontaneous idiopathic (Generally secondary to erosion of covering—pleura—of lungs due, usually, to tuberculosis)

Familial occurrence has been recorded several times.

Tuberculosis, hereditary disposition to

Depends possibly on the presence of a pair of genes; when these are lacking there is good natural resistance.

METABOLIC DISORDERS

Alkaptonuria (Congenital disorder of protein metabolism, with excretion of alkapton in urine)

R in most cases, sometimes D

Twice as frequent in males as in females.

Cystinuria (Disorder of cystine elaboration)

(i) *Childhood type* (Retention of cystine in body and excretion in urine, developmental and growth failure of bone, and degenerative lesions of kidneys, with death from uremia in many cases). A variety of genotypes have been described.

(ii) *Adult type* (May pass unnoticed or result in urinary calculi). A variety of genotypes have been described.

Fructosuria (Laevulosuria) (Defective conversion of fructose into glycogen, benign)

Probably R

Incidence: 1 in 130,000

Galactosuria (Galactosemia) (Defective conversion of galactose—a component of milk sugar—into glycogen; disorder of infants. Severe symptoms develop unless lactose eliminated from diet.)

Probably R

Glycosuria (Renal diabetes) (Sugar excreted in urine without presence in

blood to any abnormal extent, resistant to insulin; benign, no subjective symptoms)

D

Gout (Gouty arthritis) (Disturbance in purine metabolism)

Incomplete sex-limited D

90-95 percent of sufferers from gout are males.

Incidence: 88 in 100,000

Hemochromatosis (Pigmentary cirrhosis, Bronze diabetes) (Enlargement of spleen and liver with cirrhosis, slate-blue-gray color of skin, diabetes, and cardiac disease, with enormous accumulation in tissues of iron containing pigment)

Probably R

20 males to every 1 female

Hepatolenticular degeneration (Wilson's disease) (Tremor, cirrhosis of liver, eye changes, emotional lability, greenish pigment on undersurface of cornea, moderate degree of intellectual impairment, excessive secretion of amino acids in urine suggesting metabolic defect of kidney)

R

Onset: In second decade

Hyperlipemia, familial (Defective clearing of fat from the blood, associated with early onset of atherosclerosis, a senile type of arteriosclerosis)

D with incomplete penetrance

Onset: From late second decade

Incidence: 2.5 in 100

Hypoglycemia, idiopathic spontaneous (Low blood sugar with tremors, spasms, sweating, sometimes convulsions, cross-eyes, and mental retardation)

R

Lipoidosis (Lipid proteinosis) (Nodular hard lesions distributed over the head and neck and extremities; the mucous membranes of the lips, mouth, pharynx, and larynx show yellow-white patches consisting of fatty deposits. These may so narrow the larynx that the child is unable to breathe, and dies

unless a tracheotomy is performed.)

R

Lipomatosis, multiple (Overgrowth of fatty tissue, may occur in any part of body)

Probably D

Methemoglobinemia (Presence of the transformation product of oxyhemoglobin, methemoglobin, in the blood)

Sometimes D, and in some families

R

Oxalosis, familial (Deposition of calcium oxalate crystals in renal tubules and interstitial tissue, with extensive destruction and renal failure terminating in death)

D

Onset: May occur in infancy, but usually in second decade. Many more males affected than females.

Oxaluria (Abnormal excretion of oxalates in urine)

Sex-limited D

Manifests itself exclusively in males

Pentosuria (Excretion of pentose type of sugar, benign, often mistaken for diabetes)

D and R genotypes have been described.

Twice as many males affected as females

Incidence: 1 in 50,000

Phenylketonuria (Phenylpyruvic oligophrenia) (Failure of transformation of phenylalanine into tyrosine, with elimination of phenylpyruvic acid in urine—never found in normal individuals—with mental retardation and pigmentary and constitutional peculiarities)

R

Incidence: 1 in 25,000

60 percent are idiots, 30 percent imbeciles, remainder of somewhat higher mentality, epileptiform seizures frequent.

Porphyrria (Disturbance of pigment metabolism, with excretion of porphyrins and reddish or brown coloration of teeth)

- (i) *Congenital*. Probably R
- (ii) *Acute intermittent*. Irregular D

NERVOUS SYSTEM

Neurological Disorders

Acoustic neuroma, bilateral (Tumor of acoustic nerve)

D

Regarded as a form of neurofibromatosis, or von Recklinghausen's disease.

Anencephaly (Absence of the brain)

Possibly R

Occurs about one third more frequently in females.

Incidence: 1 in 1,000

Risk: There is a 2-7 percent risk that later pregnancy will result in child with defects of nervous system. There is a 20 percent chance of aborting.

Auditory imperception, congenital (Inability to understand meaning of sounds without affection of hearing)

D

Cerebral sclerosis (Schilder's disease, diffuse cerebral sclerosis, encephalitis periaxialis diffusa, subcortical encephalopathy) (Hardening of the brain)

Frequently R, but other genotypes have been described.

Disseminated sclerosis (Patches of hardening in brain, causing more or less paralysis, tremor, nystagmus, disturbances of speech, etc.)

D, with low penetrance; other forms of inheritance possible.

Incidence: 1 in 2,500

In the majority of cases if one of a pair of identical twins is affected the other is not.

Epilepsy (Chronic nervous disorder characterized by periodic convulsive attacks)

Genetic factors undoubtedly present, but at present not understood. R and irregular D cases have been described, but there

are many other genotypic patterns involved.

Incidence: 1 in 250

Hemangioma of cerebellum and retina (Lindau's disease) (Blood-vessel tumors and cysts of "little brain"—cerebellum—and inner cell layer of eyeball)

Irregular D

Huntington's chorea (Progressive muscular spasm with increasing mental deterioration)

D

Onset: At about 35 years on the average, earlier onset in females than in males.

Migraine (Visual hallucinations followed by unilateral headache and vomiting)

D

Neurofibromatosis (von Recklinghausen's disease) (Multiple tumors of nerve sheaths situated over the surface of the skin, a benign but disfiguring disease)

D, with high penetrance but variable expressivity

Pelizaeus-Merzbacher disease (Beginning in infancy, progressive general spastic paralysis, head tremor, and mental deterioration, death in third or fourth decades)

Sex-linked R, also D with diminished penetrance in female

Considerably more frequent in males

Incidence: Rare

Pick's disease (Pick's lobar atrophy) (Progressive degeneration of brain)

D

Onset: At 39-55 years

Specific dyslexia (Congenital word blindness)

D, with high penetrance

Males much more frequently affected than females

Incidence: 5 in 100.

Sydenham's chorea (St. Vitus's Dance) (Involuntary movements of limb or facial muscles)

R

Onset: In childhood

Females affected three times more frequently than males

Nerves, Muscle, and Organ Changes

Arthralgia, periodic (Joint pains at periodic intervals)

D, with high penetrance and minimal variability of expression

Bonnevie-Ullrich syndrome (Pterygium syndrome) (Abducens and facial paralysis combined with anomalies of ear, ocular adnexa, muscles of limbs, especially hands)

Irregular D

Cerebral diplegia, congenital (Spastic weakness of legs at birth, sometimes with ataxia and mental defect from birth, with tendency to improvement)

Probably one or more R genes

Family periodic paralysis (Periodic production of muscular weakness or paralysis)

Has been reported as D, R, and sex-linked R

Hallervorden and Spatz syndrome (Increasing rigidity of limbs with difficulty of speech and progressive dementia)

R

Heredopathia atactica polyneuritiformis (Refsum's disease) (Night blindness, atypical retinitis pigmentosa, later polyneuritis, and muscular weakness, terminated by sudden death)

R

Onset: In the second to fourth decades

Muscular dystrophy, progressive

(i) *Facioscapulohumeral type*. D.

Onset: Usually between 7 and 20 years

(ii) *Childhood progressive muscular dystrophy (pseudohypertrophic type)*. Sex-linked R. Onset: Usually at about 3rd year; invalids by 9-12 years; death often before 25 from respiratory infections. Mutation rate: About 1 in 10,000 male births

Muscular weakness, atrophy, and mental deficiency

Sex-linked R

Myotonia congenita (Thomsen's disease) (Muscle spasm or rigidity)

D, with reduced penetrance in some families

Myotonia dystrophica (Extreme weakness, usually of muscles of face and neck, which may spread to other parts of body)

Irregular D

Cataract common in otherwise normal parents and relatives, as well as tendency to baldness

Paralysis agitans (Shaking palsy)

D, with penetrance of about 60 percent

Paramyotonia (Muscular spasm or rigidity in reaction to cold)

D

Peroneal muscular atrophy (Charcot-Marie-Tooth's disease) (Degeneration of muscles of legs, feet, and hands)

D, R, and sex-linked genotypes have been described.

Onset: Neither parent affected, at 11 years; one parent affected, at 19 years

Progressive hypertrophic polyneuritis (Shooting pains and numbness in extremities, muscular weakness and atrophy in hands, forearms, and legs)

D

Spastic diplegia with idiocy or imbecility, congenital (Diplegia-bilateral paralysis)

Probably R

Incidence: 1 in 1,000-2,000 live births

Onset: At end of first year

Spastic paraplegia (Spasmodic paralysis of the lower extremities)

D, R, and sex-linked genotypes have been described; multiple alleles or more than one gene pair may be involved.

More frequent in males

Spinocerebellar ataxia (Loss of muscular co-ordination)

- (i) *Friedrich's ataxia*. Usually R
 - (ii) *Marie's ataxia*. Usually D
- Several alleles or more than one pair of genes may be involved in these and other forms of hereditary ataxia.

Torsion dystonia (Turning and twisting movements of trunk and proximal parts of limbs, with loss of muscle tone)

R

Mental Deficiencies

Amaurotic idiocy, familial (Abnormal storage of fats, mental impairment leading to idiocy, progressive blindness, paralysis, wasting, and death)

- (i) *Infantile type (Tay-Sachs)*. R. Incidence: 1 in 250,000. Onset: At 1-6 months, death within 2 years.
- (ii) *Juvenile type*. R. Incidence: 1 in 40,000. Onset: At about 6 years, death at 15-20
- (iii) *Adult type*.

Cataract with idiocy or imbecility, congenital

Probably R

Incidence: 1 in 50,000 live births

Risk for siblings: 16 percent

Cretinism (Congenital arrest of mental and bodily growth at infantile level)

The type that does not respond to thyroid treatment is probably genetically determined, but in a manner unknown.

Epiloia (Bourneville's disease) (Tumors of brain, skin, and viscera associated with mental defect in 70 percent of cases, with epilepsy in 80 percent)

Irregular D

Incidence: 1 in 50,000

Feeble-mindedness (I.Q. range 50-69)

Many different genotypes involved

Idiocy (I.Q. range, 0-19)

Apparently many different genotypes involved

Imbecility (I.Q. range, 20-49)

Many different genotypes involved

Laurence-Moon-Bardet-Biedl syndrome (Underdevelopment of external genitalia, obesity, retinitis pigmentosa, polydactyly, and mental deficiency)

R, with variable penetrance and expressivity

Almost twice as frequent in males as in females

Microcephaly, familial (Familial "pin-headism")

Probably R

Incidence: 1 in 25,000-50,000 births

To be distinguished from forms of microcephaly that are caused by irradiation *in utero*, toxoplasmosis, and similar environmental conditions

Microphthalmos and mental deficiency (Small eyes with mental deficiency)

Sex-linked R

Incidence: 2.5 in 100,000

Risk: With both parents unaffected where a child is affected, for sibs, 9 percent; in those cases in which the microphthalmic child is mentally normal, risk for future siblings very low.

Mongolism (Mongolian idiocy)

Characterized by 47 chromosomes instead of 46, but other genetic factors almost certainly involved but not understood.

Incidence: 1 in 700 births

Risk: Increases with increasing age of mother. From the age of 40 the chance of having a mongoloid child is from 1 to 6 percent. Risk of recurrence is low.

Mental Disorders

Autonomic dysfunction (Riley's syndrome) (Crying without tears, excessive sweating, skin blotches, emotional instability, motor incoordination, etc.)

Probably R

Manic-depressive psychosis (Mental disorder characterized by marked emotional oscillation between manic and depressed states, usually cyclical)

D, with incomplete penetrance
Incidence: Slightly less than 1 per cent

Neurotic temperament (Functional nervous disorder)

In many cases largely environmentally caused, in still others probably a condition involving a number of genes which will express themselves only in the appropriate environment.

Schizophrenia (Disturbance in reality relationships, fragmented personality)

Some R, but with variable expressivity; others D, with variable expressivity; some cases not genetic at all

Incidence: 1 in 100 to 1 in 200.

NOSE

Anosmia (Total absence of sense of smell)

D

Choanal atresia (Occlusion of the openings of the nasal fossae into the pharynx)

Probably D

Epistaxis (Nosebleeding, often due to varicose enlargement of nasal veins)

Probably D

"Potato nose" (Upper part of nose distended like a balloon)

Probably irregular D

Rhinitis (Inflammation of nasal mucous membrane)

(i) *Acute catarrhal rhinitis* (In childhood). D

(ii) *Chronic hypertrophic rhinitis* (Chronic catarrh). D

(iii) *Atrophic rhinitis* (Chronic catarrh with thinning of mucous membrane). D

(iv) *Polyposis* (Polyps of nose and sinuses). Genetic mechanism unclear

Rhinophyma (*rosacea of the nose*) (Reddish-blue, swollen, large dilated pores of lower part of nose)

Has been observed in over three

generations; genetic mechanism not clear

SKIN

Acanthosis nigricans (Hypertrophy with pigmentation of papillae of outer layer of skin)

(i) *Benign*. D and irregular D

(ii) *Malignant*. Possibly R

(iii) *Pseudoacanthosis with obesity*. Probably environmentally conditioned

Albinism (Congenital absence of pigment in skin and its appendages)

(i) *Generalized* (Albinism of the whole body). R. Also occurs as D. Incidence: 1 in 15,000–20,000

(ii) *Partial* (Albinism of forehead, neck, linea alba, or white forelock). D

(iii) *Ocular* (Albinism limited to eye). Sex-linked R

Eczema (Inflammation of the skin with multiple lesions)

Irregular D

Epidermodysplasia verruciformis (*Lewandowsky-Lutz disease*) (Flat polygonal papules first appearing on limbs and then spreading to face, hands, and feet, and occasionally to whole body)

Probably R

Epidermolysis bullosa (*Goldscheider's disease*) (Blistering of the skin with generalized pathology)

(i) *Simplex type*. D, with reduced penetrance. 59 percent of those affected are males. Onset: In first year

(ii) *Dystrophic hyperplastic type*. D, with high penetrance. Onset: Any time between birth and puberty

(iii) *Dystrophic hypoplastic type*. R, occasionally incompletely sex-linked R. Onset: Usually soon after birth

Erythema nodosum, familial (Hypersensitive skin reactions due to allergens associated with many diseases, with

- accompanying constitutional upset)
Probably D with variable penetrance
- Hyperelastosis cutis* (Ehlers-Danlos syndrome) (Extreme laxity of skin and joints and fragility of skin and blood vessels)
D, with variable penetrance and expressivity
Somewhat more frequent in males
- Ichthyosis* (Horny plating and fissuring of skin)
(i) *Ichthyosis fetalis*. R. Death at birth or shortly afterward
(ii) *Ichthyosis congenita*. R
(iii) *Ichthyosis vulgaris*. D, with variable expression and manifestations; sometimes sex-linked R
- Keloids, multiple* (Fibrous tumors of the skin)
D
Onset: Any age, usually at end of second decade. Occurs about 9 times more frequently in Negroes than in whites.
- Keratosis*
(i) *Keratosis follicularis* (Darier's disease) (Numerous follicular papules over body). D
(ii) *Keratosis follicularis spinulosa* (A mild form of keratosis follicularis). Sex-linked R
- Nevi* (Birthmarks)
D where due to heredity
- Pachyonychia congenita* (Heaped-up, clawlike nails, thickening of skin, excessive sweating, etc.)
D
Males affected 4 times more frequently than females
- Piebaldness* (Forehead blaze with non-pigmented spotting on limbs and trunk)
Usually D
- Pityriasis rubra pilaris* (Papular disease of skin with scaling)
D, with high penetrance
- Porokeratosis* (Keratinization, or horniness, of skin)
D
- Males affected twice as frequently as females
- Pseudoxanthoma elasticum* (Grönblad-Strandberg syndrome) (Papular eruption, usually on neck and in armpits, but may appear anywhere on body, associated with blood streaks in retina)
Probably R, with partial sex-limitation to female
- Psoriasis* (Chronic red-brown, scaly papules of skin)
Irregular D, sometimes R
Risk: For siblings of affected child, parents normal, 2 to 7 percent; one affected parent, 9 to 20 percent. For children of affected parent, 15 percent.
- Recurrent bullous eruption of feet* (Blistering of soles of feet, particularly in hot weather)
D
Preponderance of males
- Tylosis* (Thickening of skin of palms, soles, and flexor surfaces of digits)
D, with high penetrance
Onset: May start at birth or be delayed for 5 or 6 years
- Urticaria pigmentosa* (Red-brown pigmented macules, or blemishes, of skin)
Possibly R
- Xanthelasma* (Fatty skin growths, usually on eyelids)
D
- Xanthomatosis*
(i) *Tuberous* (Yellow nodules in skin). D
(ii) *Coronary* (Yellow nodules in heart). D
(iii) *Familial hypercholesteremia* (Yellow nodules in skin associated with high cholesterol concentrations in blood. Nodules occur also on periosteum and tendons). D
- Xeroderma pigmentosum with multiple basal-cell cancer* (Freckles, warty growths, superficial ulcers, and malignant changes; earliest signs reddening and excessive freckling during first

summer of infant's life; death generally before 30)

Incompletely sex-linked R

TEETH AND MOUTH

Ankyloglossia (tongue-tie)

D

Caries (Tooth decay)

Probably D

Central Incisors, absence of all

Incomplete D

Central Incisors, very large

D

Chin, receding (Angle's Class II deformity)

Irregular D

Cleft palate (without harelip)

D with variable penetrance

Many more females than males affected.

Incidence 1 in 2,500

Risk: Normal parents with no affected relatives, 1 in 80; with both parents normal and an affected relative, about 1 in 8; with one parent affected, about 1 in 6; children of an affected parent, married to a normal, about 1 in 15

Dentin dysplasia (Rootless teeth)

D

Dentinogenesis imperfecta (Imperfect formation of dentin)

D

Incidence about 1 in 8,000

Enamel, defective development of

- (i) *Agensis* (only thin covering of enamel present, molars may fail to develop). Sex-linked D
- (ii) *Hypocalcification* (Loss of enamel shortly after eruption of teeth.) D
- (iii) *Hypomaturation* (Soft enamel) Sex-linked R
- (iv) *Pigmented Hypomaturation* (Soft pigmented enamel) Probably R
- (v) *Local Hypoplasia* (Brown or

lined undeveloped areas, mostly on incisors and premolars. Milk teeth alone may be affected.)

D with incomplete penetrance

Gingival hyperplasia (Overgrowth of gums)

D

Harelip (with or without cleft palate)

R, sex-linked, and incomplete D genotypes have been described.

Twice as many males affected as females.

Incidence: 1 in 1,000

Risk: When both parents normal, 1 in 20; for siblings when one parent affected, 1 in 10; children of an affected parent, married to a normal, 1 in 50

Jaw, fibrous swelling of

Possibly D

Jaw, protrusion of lower (Angle's Class III deformity)

D

Lateral Incisors, absence of

D

Molars, absence of

D

Premolars, absence of

D

Teeth, absence of all

D

Teeth, supernumerary

D

MISCELLANEOUS

Ectodermal dysplasia

- (i) *Anidrotic type* (Absence of sweat glands, peculiarities of skin, and developmental failure of teeth to complete absence of teeth). Usually sex-linked R
- (ii) *Idrotic type* (Sweat glands normal, other symptoms present). Usually D

Epithelioma adenoides cysticum (Multiple benign cystic adenomas)

D, with partial sex limitation to females

Gargoylism (Hurler-Pfaundler syndrome, dysostosis multiplex) (Dwarfism, enlarged liver and spleen, corneal opacities, and mental deficiency; death from heart failure usually before 20)

Usually R. The less severe form, without clouding of the cornea, indicates sex-linked R inheritance.

Gaucher's disease (Disorder of the reticulo-endothelial system—spleen, liver, lymph nodes, bone marrow)

(i) *Infantile type*. Probably R. Onset: Acute, at about 6th month; ends in early death

(ii) *Adult type* (Benign). Probably D. Onset: In advanced age

Laryngitis

(i) *Acute*

(ii) *Chronic*

That there is a hereditary tendency involved in many cases of laryngitis has long been known, but the precise genetic mechanism is not understood.

Leprosy (Hansen's disease)

Probably irregularly D

Lymphedema, congenital (Milroy's disease) (Disorder of the lymph vessels, usually confined to lower limbs)

D, with low penetrance

Preponderance of females and female transmitters

Marcus Gunn phenomenon (Ptosis, winking when jaw moves)

Irregular D

Pancreatic cystic fibrosis (Involvement of the pancreas, small intestine, and bronchioles of lung)

R

Incidence: 1 in 1,000

Risk: For parents having one or more affected children, 1 in 4

Rheumatic fever

The predisposition may be inherited as a R which will find expression only under the appropriate environmental conditions.

The Chance of the Offspring of Marriage Between Cousins Exhibiting Defective Condition

IT HAS BEEN estimated by Muller (1950) that the average person carries about eight deleterious mutations in the recessive state. In a random system of mating it is unlikely that husband and wife would also carry any one of these same recessive genes; each would carry different recessive genes. When second cousins marry there is 1 chance in 16 that both would be carrying one of the sixteen recessives. Second cousins would be the grandchildren of independent sets of grandparents, one member of each grandparental set standing in the relation of brother or sister to each other. The chance that both the intermarrying grandchildren would not carry the same recessive gene is $\frac{15}{16}$, and the chance that both of them would not carry the same one of any of the sixteen recessive genes originally carried by the grandparents between them is $(\frac{15}{16})^{16}$ or 0.356. The probability, then, that any two of the grandchildren would carry any one of the sixteen recessive genes shared between the grandparents is equal to $1 - (\frac{15}{16})^{16}$ or 0.644. The probability that the first child of the union between such grandchildren would be homozygous for any one of the recessives carried by the parents is one fourth of 0.644 or 0.161. Should the offspring of such unions amount to four individuals, then 0.644 of these families, on the average, would have at least one individual who would be homozygous for a deleterious recessive. In other words, nearly 65 percent of such families would have at least one member who was in some way unfavorably affected by the defective recessive genes carried in him in the homozygous state.

However, the truth is that not all eight deleterious genes are necessarily related to congenital or childhood abnormalities. The mutant genes involved may exert their effect at any time between conception until terminal age, say up to a hundred years or more. If, then, with Sheldon Reed, who has considered this problem in relation to the practical exigencies of counseling, we arbitrarily assume that one fifth of the sixteen deleterious genes of the grandparents may produce congenital or childhood abnormalities, one fifth of 0.161 or 0.032 (1 in 31) of the cousin marriages should result in a congenital or childhood abnormality in the first child, or 1 in 8 of cousin marriages should result in such an anomaly where at least four children are born as the result of such a cousin union.

Location, Name of Institution, and Principal Counselor of Some Heredity Clinics (By State)

- Berkeley, California. Department of Zoology, University of California, Curt Stern.
- Chicago, Illinois. Department of Zoology, University of Chicago, H. H. Strandskov.
- New Orleans, Louisiana. The Medical School, Tulane University, H. W. Kloepfer.
- Ann Arbor, Michigan. Department of Medical Genetics, University of Michigan, J. V. Neel.
- Minneapolis, Minnesota. Dight Institute, University of Minnesota, S. C. Reed.
- Rochester, Minnesota. The Mayo Clinic, J. S. Pearson.
- New York, New York. New York State Psychiatric Institute, F. J. Kallmann.
- Winston-Salem, North Carolina. Department of Preventive Medicine, Bowman Gray School of Medicine, C. N. Herndon.
- Cleveland, Ohio. Department of Zoology, Western Reserve University, A. G. Steinberg.
- Columbus, Ohio. Starling Loving Hall, Ohio State University, M. T. Macklin.
- Norman, Oklahoma. Department of Zoology, University of Oklahoma, P. R. David.
- Providence, Rhode Island. Institute for Research in Health Services, Brown University, G. W. Hagy.
- Austin, Texas. The Genetics Foundation, University of Texas, C. P. Oliver.
- Salt Lake City, Utah. Laboratory of Human Genetics, University of Utah, C. M. Woolf.
- Charlottesville, Virginia. School of Medicine, University of Virginia, R. F. Shaw.

Richmond, Virginia. Department of Biology and Genetics, Medical College of Virginia, B. L. Hanna.

Seattle, Washington. School of Medicine, University of Washington, A. G. Motulsky.

Madison, Wisconsin. Department of Medical Genetics, The Medical School, University of Wisconsin, N. E. Morton.

Toronto, Ontario. Hospital for Sick Children, N. F. Walker.

Montreal, Quebec. Department of Genetics, McGill University, F. C. Fraser.

Glossary

Acquired character. A character which is the result of the environment. Acquired characters are *not* inherited.

Adaptation. The process, and the result of the process, by which members of a genetic group, either as individuals or in part or as groups, are fitted to past or present changes in their environment. An adaptation is any trait of the organism that contributes to its survival or the survival of the group of which it is a member in the environment which it inhabits.

Allele or Allelomorph. Any of the various forms of a gene. Alleles occupy the same position (*locus*) on a given chromosome, influencing in different ways the same developmental process.

Amino acid. One of a number of organic acids containing the amino radical (NH_2), from which proteins are formed.

Anaphase. The stage of mitosis (or meiosis) following metaphase when the daughter chromosomes are separating toward the poles of the spindle.

Androgen. A hormone, secreted in both sexes, but more abundantly in males, that influences the development of maleness (in structure, form, and behavior). The principal male sex hormone is testosterone which is of testicular origin. Androsterone is another male sex hormone.

Anthropometry. The measurement of the human body by weight, size, and proportions.

Antibody. One of certain chemical substances formed by the body when a foreign material is introduced into the blood stream. The antibody usually combats the ill effects of the foreign material.

Asexual. Designating any mode of reproduction that does not involve the union of male and female reproductive cells.

Atavism. The wholly erroneous notion that long extinct ancestral traits can reappear in a descendant.

Autosome. Any of the twenty-two pairs of chromosomes which are not sex chromosomes. All human beings carry twenty-two pairs of

- autosomes and one pair of sex chromosomes. The abnormal exceptions may carry more or less.
- Backcross.* The cross of a hybrid with one of its parents.
- Bivalent.* A pair of homologous chromosomes united at the first meiotic division, usually by chiasmata.
- Catalyst.* A substance that causes or hastens a chemical change without itself being changed. Genes are autocatalytic.
- Caucasoid.* One of the major groups of mankind, or a member of that group, characterized most generally by white skin (though many Caucasoids have dark skins), a long nose, and straight or curly hair.
- Cell.* The living active unit of all plants and animals, consisting of many specialized parts. In the nucleus of the cell lie the chromosomes.
- Cell differentiation.* Specialization of the cells for different kinds of function, differing from ordinary growth (increase in size) in that the cells become unlike the parent cells.
- Centriole.* A small body in the centrosome situated just outside the nuclear membrane. The centriole organizes both the spindle and the centromere for movement.
- Centromere.* The small body that holds together at their middle the double strands forming each chromosome: best seen in prophase. In metaphase the centromeres split into two, thus entering upon anaphase, when each of the centromeres with its chromosome becomes attached to a spindle, the centromeres finally moving apart to attach one to each centriole.
- Centrosome.* The self-propagating body which during mitosis divides into two parts, each lying at either pole of the spindle. The centrosome appears to determine the formation of the spindle.
- Character.* A property of an organism in regard to which observations of genetic or other similarities or differences are made. Also called *trait*.
- Chiasma.* A crosswise exchange of partners in a system of paired chromatids observed just before the beginning of anaphase in meiosis. Plural, *chiasmata*.
- Chromatid.* A longitudinal half chromosome between early prophase and metaphase of mitosis, and between diplotene and second metaphase of meiosis.
- Chromosome.* One of a number of thread-shaped bodies situated in the nucleus of animal and plant cells, and carrying the genes. Usually visible only during mitosis or meiosis.
- Conceptus.* The organism from conception to birth.
- Concordant.* Agreeing in traits: used mostly in twin studies.
- Congenital.* Existing at birth as a result either of one's genotype, or of one's prenatal environment.
- Corpus luteum.* The yellowish body on the surface of the ovary mark-

- ing the place through which a ripe ovum has passed. The corpus luteum functions as a temporary gland secreting progesterone, but only under the influence of the organizing action of the pituitary hormone luteotrophin. See *progesterone*.
- Cortex*. The outer layers of an organ, in contrast to its inner substance, the *medulla*.
- Cross*. A mating between two individuals.
- Crossing over*. The exchange of material between homologous chromosomes at meiosis, containing corresponding genes at the same loci as were present on each homologous chromosome. Hence, the shifting of genes from one chromosome to another.
- Culture*. That part of the environment which is learned; the man-made part of the environment. The way of life of a people.
- Cytoplasm*. All the protoplasm of a cell except that of the nucleus, which is called *nucleoplasm*.
- Deoxyribonucleic acid (DNA)*. The principal constituent of the gene, believed to be the material of heredity itself. DNA is thought to carry the master plans or code containing the information which determines the order in which the amino acids fall into place in the protein molecule for which it is responsible.
- Diploid*. Containing the chromosomes in pairs, the members of which are homologous, i.e., containing two sets of chromosomes or twice the haploid number: characteristic of all the cells of the body except the gametes.
- Diplotene*. The stage of meiosis following the division of the chromosomes and chiasma formation at pachytene.
- Discordant*. Having a different trait or traits: used mainly in twin studies.
- Disease*. An acquired morbid change in any tissue or tissues of an organism, or in an organism as a whole, of specific microorganismal causation with characteristic symptoms. See *disorder*.
- Disorder*. A disturbance of structure or function or both due to a genetic or embryological failure in development or as the result of exogenous factors, such as certain chemical substances, injury, or disease. May be inborn or acquired. See *disease*.
- Dizygotic*. Derived from two fertilized eggs (zygotes).
- Dominant gene*. A gene which in a heterozygote overrides the effect of its recessive allele. A gene that always expresses itself in the individual who possesses it.
- Drosophila*. The generic name of the banana fly, vinegar fly, or fruit fly.
- Dysfunction*. Abnormal or incomplete functioning of an organ.
- Embryo*. An animal in process of development from the zygote. From the time of conception to the end of the second week of development, the human organism is sometimes said to be in the *ovular* phase. From the end of the second week to the beginning of the

- eighth week, it is in the *embryonal* phase, and from the beginning of the third month to birth, it is in the *fetal* phase.
- Embryology*. The study of the early phases of development of an organism.
- Endocrine gland*. Any of certain glands of internal secretion, that secrete directly into the blood stream and not through ducts, hence, also called *ductless gland*.
- Environment*. The external conditions which are acting upon or have acted upon an organism. The interaction with the genotype determines the phenotype.
- Enzyme*. Any of various organic compounds in the body that act as catalysts, causing the chemical processes of the body to be carried on.
- Epicanthic fold*. A fold of skin from the upper eyelid lying over the inner angle of the eye or extending over the whole of the upper eyelid. Sometimes called the "Mongoloid fold."
- Epigenesis*. The obsolete theory that the germ cell is structureless and that the embryo develops as a new creation through the action of the environment. Opposed to *preformation*.
- Epistasis*. The suppression of the visible action of one gene by another that is not its allele.
- Estrogen*. Any of the female sex hormones, so called because they are capable of inducing estrus in the female, whether mature or immature, spayed or not. The estrogens are *estrone*, *estriol*, and *estradiol*.
- Estrus*. The period of physiological changes in the reproductive organs of the female when in heat.
- Ethnic group*. An arbitrarily recognized population which, having a more or less distinctive assemblage of physical traits, through a common heredity, is distinguishable from other populations within the species.
- Eugenics*. The science of the improvement of human fitness and the reduction of unfitness through the control of heredity.
- Expectation of life*. A measure or statement of the number of years an individual may, on the average, expect to live, from birth or from any age taken as a datum point. It is computed on the basis of the actual ages at death of the population in any given age range at any given age. Often used roughly in the same sense as *longevity*.
- Expressivity*. The manifestation of a trait produced by a gene. When the manifestation differs from individual to individual, the gene is said to have *variable expressivity*, for example, the dominant gene for allergy may take such forms as asthma, eczema, hay fever, angioneurotic edema, or urticarial rash.
- Fertilization*. The union of egg and sperm.
- Fetus*. The intrauterine organism from the beginning of the third month to birth. See *embryo*.
- Fraternal twins*. Twins developed

from separate eggs; two-egg twins or dizygotic twins.

Gamete. A mature germ cell; the *ovum* of the female or the *spermatozoon* of the male.

Gene. The physical unit of heredity, a giant molecule believed to consist mainly of deoxyribonucleic acid (DNA). It is estimated that man has a total of about 30,000 genes on his 46 chromosomes.

Gene dosage. The number of times a given gene is present in the nucleus of a cell or organism.

Genetic Drift (The Sewall Wright Effect). The nonselective random distribution, extinction, or fixation of genes in a population.

Genetic equilibrium. The condition of a population in which successive generations consist of the same genotypes with the same frequencies, in respect to particular genes or arrangements of genes.

Genetics. The branch of biology concerned with the manner in which inherited differences and resemblances come into being between similar organisms.

Genotype. The genetic constitution, determined by the number, types, and arrangement of genes.

Germ plasm. The hereditary material present in germ cells.

Glutamic acid. An amino acid occurring as a decomposition product of protein.

Gonad. The sex gland which produces gametes; the *ovary* in the female or the *testis* in the male.

Haploid. Containing a single set of

unpaired chromosomes: characteristic of gametes.

Heterogametic sex. The sex which forms two types of gametes in equal proportions; the male, because half the spermatozoa carry an X and half carry a Y chromosome in addition to the autosomes.

Heterozygous. Carrying different alleles of a gene on both homologous chromosomes.

Homogametic sex. The sex which forms only one type of gamete; the female, because all the ova carry only the X chromosome in addition to the autosomes.

Homologous chromosomes. Partner chromosomes, usually identical in form and in the number and types of genes they contain. The exceptions are the sex chromosomes, X and Y, which differ from one another in form and gene content.

Homozygous. Carrying the same alleles of a gene on both homologous chromosomes.

Hormone. A chemical substance produced in small quantity in an organ and transported to other parts where it exerts specific effects.

Hybrid. The offspring of two genetically different parents. Commonly, the offspring of parents of different ethnic or racial origin.

Hybrid vigor. Increased vigor of growth, fertility, and other traits in a hybrid of two different stocks, as compared with either of the parental lines.

Hydrocephalus. A condition characterized by great enlargement of

the head, generally as a result of increased fluid in the lateral ventricles of the brain. The condition is usually congenital.

Identical twins. Twins that develop from the same egg; one-egg twins or monozygotic twins.

Immunity. The capacity to resist, or freedom from susceptibility to, disease-producing organisms or poisons.

Incomplete dominance. The relationship between two alleles which in the heterozygote produces an intermediate effect. Negroid skin color genes are incompletely dominant.

Intelligence. The individual's total repertory of those problem-solving and thinking discriminatory responses that are usual and expected at a given age level. The "usual and expected" response is one of which 65 to 75 percent of the given population is capable.

Isolation. The condition in which potential mating groups are separated by ecological or social barriers and thus prevented from mating.

Lethal gene. A mutant gene, the effect of which is to kill an organism at any stage from egg to adult.

Linkage. Genes situated on the same chromosome are said to be linked. *Close linkage* refers to the association of non-allelic genes which act as if they were inseparable, usually because they are located at or near the same locus

on the same chromosome. The linkage is broken by crossing over.

Locus. A particular place in a particular chromosome which always contains one kind of gene, or one of a particular set of alleles. Homologous chromosomes usually have identical sets of loci.

Longevity. The span of years lived. Often used roughly in the same sense as *expectation of life*.

Maturation division. See *meiosis*.

Medulla. The inner substance of an organ in contrast to its outer substance, the *cortex*.

Meiosis. The two successive cell divisions, from a diploid mother cell, preceding formation of the gametes. Both divisions resemble mitosis, except that while there are two divisions of the nucleus, in the second meiotic division the chromosomes are not duplicated, hence resulting in the formation of haploid gametes from diploid mother cells. See also *mitosis*.

Melanin. A complex, dark brown pigment which, in various concentrations, affects the color of hair, skin, and eyes.

Menarche. The first menstruation.

Mendel's laws. The three principles of chromosome behavior at meiosis: 1. *The Law of Segregation* refers to the behavior of one pair of genes. When the homologous chromosomes separate, the two alleles segregate into different gametes. 2. *The Law of Independent Assortment* or *Free Recombination* refers to the behavior of

two or more pairs of alleles carried on different chromosomes. Each pair of chromosomes separates into different gametes independently of other pairs. The result is that the gametes contain all possible combinations of the genes constituting the different pairs. *Linked genes* are the exception to this law; recombination of such genes occurs through *crossing over*. 3. *The Law of Dominance and Recession* refers to the fact that some genes are capable of suppressing the expression of other allelic genes. Genes of the first class are called *dominant*, genes whose expression is suppressed are called *recessive*. Recessive genes do not or only minimally express themselves in heterozygous condition, but *do* fully express themselves in homozygous condition.

Metabolism. The building up or breaking down of protoplasm within an organism. E. B. Wilson wrote in 1896 that "inheritance is the recurrence, in successive generations, of like forms of metabolism." More generally, the sum of the chemical changes whereby nutrition is effected.

Metaphase. The stage following prophase in mitosis (or meiosis) when the chromosomes arrange themselves at the equator of the spindle.

Mitosis. A process of cell division during which the genes and chromosomes reduplicate (*prophase*), migrate to an equatorial plane (*metaphase*), and separate to opposite poles (*anaphase*); the cell

splits and forms two new cells (*telophase*). See also *meiosis*.

Modification. A nonheritable change.

Modifier. A gene which modifies the effects of the phenotype controlled by one or more pairs of genes.

Molecule. The smallest possible unit of any substance that can exist and in the free state retain the characteristics of that substance. The molecules of elements consist of one atom or two or more similar atoms; those of compounds consist of two or more different atoms.

Mongolism or *Mongolian idiocy*. A developmental disorder, associated with a genetic constitution in which there are forty-seven instead of forty-six chromosomes, occurring significantly more frequently in children of mothers who were more than thirty-five years when they conceived. Mongolism is characterized by severe mental and physical retardation, the presence of epicanthic folds, a large space between the first and second digits of the hands and feet, stubby hands and fingers, a large tongue, etc.

Mongoloid. One of the major groups of mankind, or a member of that group, generally characterized by a flattened face, high cheekbones, marked overbite of upper teeth, shovel-shaped incisor teeth, epicanthic folds, very slight yellowish tinge to the skin, and lank black hair.

Mongoloid fold. See *epicanthic fold*.

Monozygotic. Derived from one fertilized egg (zygote).

Multiple alleles. A series of three or more alleles of one gene.

Multiple factors. See *polygenes*.

Mutation. A failure of precision in the basic property of self-copying in a gene, resulting in a transmissible hereditary modification in the expression of a trait. The effects of most mutations in any one generation are usually not detectable.

Mutation pressure. The measure of the action of mutation in tending to alter the frequency of a gene in a given population.

Natural Selection. A shorthand phrase for the effects of the differential reproduction of different types. Defined by Darwin: "As many more individuals of each species are born than can possibly survive; and as, consequently, there is a frequently recurring struggle for existence, it follows that any being, if it vary however slightly in any manner profitable to itself, under the complex and sometimes varying conditions of life, will have a better chance of surviving, and thus be *naturally selected*. From the strong principle of inheritance, any selected variety will tend to propagate its new and modified form." *Origin of Species*, 1859, p. 5.

Negroid. One of the major groups of mankind, or a member of that group, characterized by dark pigmented skin, kinky, fuzzy, or

woolly hair, and, generally, a broad nose.

Nondisjunction. The failure of separation of paired chromosomes at meiosis or mitosis, and their passage to the same pole. *Primary nondisjunction* is the production of eggs with two X chromosomes or none by an XX individual. *Secondary nondisjunction* is the production of eggs with two X chromosomes or a Y chromosome by an XXY individual.

Nucleoplasm. The protoplasm constituting the nucleus of a cell.

Nucleotide. Nucleic acid consisting of three components: phosphoric acid, carbohydrates, and purine and/or pyrimidine bases combined in one unit. Nucleotides are thought to exist in aggregates of four:

phosphoric acid—sugar—base

phosphoric acid—sugar—base

phosphoric acid—sugar—base

phosphoric acid—sugar—base

Observe that the asymmetric structure of the tetranucleotide conforms well with the spiral structure of the DNA molecule. Deoxyribonucleic acid (DNA) is a nucleotide. The nucleic acid may be built up out of 1,000 such tetranucleotides.

Ontogeny. The life development of a single organism.

Oöcyte. The egg maternal cell with diploid nucleus which, by meiosis, results in four haploid nuclei.

Ovulation. The discharge of the ripe egg from the ovarian follicle onto the surface of the ovary, whence the egg is then usually taken up by the fimbriated ends of the fallopian tube.

Pachytene. The double thread produced by pairing of the chromosomes in the prophase of meiosis.

Paramutation. A heritable change from one allele to another produced by a particular allele with which it is brought into association. Such paramutations are not entirely stable, and under certain conditions will revert partially, but not completely, to the former normal state.

Parity. The state of a female with regard to her having borne children. *Nulliparity* is the condition of having borne no children, *primiparity* of having borne one child, *secundiparity* two children, and *multiparity* three or more.

Parthenogenesis. Reproduction without fertilization.

Penetrance. The regularity with which a gene produces its effect. The proportion of organisms homozygous for a particular gene which show the effect of that gene. When a gene regularly produces the same effect it is said to have *complete penetrance*. When the trait is not manifested in some individuals the gene is said to have *reduced penetrance*. Dominant genes with reduced penetrance

may be mistaken for recessives. When the penetrance of an autosomal gene is completely reduced in one sex, the gene is sex limited.

Phenocopy. A nonhereditary variation or trait phenotypically indistinguishable from the hereditary one.

Phenotype. The manifest characteristics of the organism, including anatomical, physiological, and psychological traits. The product of the joint action of environment and genotype.

Pleiotropy. The effects of a gene on more than one character.

Polygenes. A series of two or more genes affecting the same phenotypic character, usually in a quantitative (additive or multiplicative) way. Also called *multiple factors*.

Polyplloid. Having more than two sets (*diploid*) of chromosomes: *triploid* (three), *tetraploid* (four), *pentaploid* (five), *hexaploid* (six), etc.

Population. Any contiguously distributed grouping of a single species which is characterized by both genetic and social continuity through one or more generations.

Position effect. The difference in effect of two or more genes according to their spatial relations in the chromosomes.

Preadaptation. Mutational or already existing genes carried in an individual or a group which enable their bearer or bearers to fill a new niche in the environment when, by chance, it is presented.

Preformation. The obsolete theory that the entire diversity of struc-

- ture is contained in the embryo, and that development consists merely in increase in size (growth). Opposed to *epigenesis*.
- Progesterone*. A hormone secreted by the corpus luteum of the ovary, and during pregnancy by the placenta. Prepares the uterus for receiving and developing the fertilized ovum.
- Prophase*. The first stage of mitosis (or meiosis) during which chromosomes appear, and in meiosis undergo pairing.
- Propositus* or *Proband*. An individual whose condition leads to the ascertaining of a pedigree.
- Protein*. Any of certain nitrogenous substances consisting of a complex union of amino acids and containing carbon, hydrogen, nitrogen, oxygen, and frequently sulfur. A chief constituent of chromosomes, and plant and animal bodies.
- Protoplasm*. The essential substance in all plant and animal cells.
- Race*. A word widely and frequently misused, hence better not used at all. By geneticists used to mean any population that differs from other populations in the frequency of its genes. By anthropologists a population whose physical characters, through a common heredity, distinguishes it from other populations. The author prefers to use the non-committal "ethnic group."
- Random mating*. Mating that is largely determined by chance, rather than by careful choice. More especially, chance mating with respect to any particular gene.
- Recessive gene*. A gene which does not show its major effect in the heterozygote.
- Selection pressure*. The measure of the action of selection in tending to alter the frequency of a gene in a given population.
- Selective advantage*. The genotypic condition of an organism or group of organisms which increases its chances, relative to others, of representation in later generations.
- Sex chromosome*. The chromosome which determines the sex of the organism. In mammals and many other organisms the female has two X chromosomes, the male has an X and a Y. In the fruit fly, *Drosophila*, sex is determined by whether or not a single or double complement of certain genes carried in the X chromosomes is balanced against certain genes carried in the autosomes. The double complement produces a female, the single complement a male. But in man and mouse, and probably all other mammals, it is the Y chromosome that carries the male-determining factors. The X chromosome probably carries some female-determining factors, while others are situated on the autosomes.
- Sex-influenced trait*. Any trait conditioned by genes carried in the autosomes, and hence inherited equally by both sexes. Sex, however, controls the dominance. The

- gene which is dominant in one sex is recessive or intermediate in the other, and vice versa.
- Sex-limited trait.* Any of certain traits expressed in one sex but not in the other, conditioned by genes carried either in autosomes or in sex chromosomes, the expression of sex-limited traits depending largely upon the presence or absence of one or more sex hormones, or, strictly speaking, upon the amount of such hormones circulating within the body of the organism.
- Sex-linked gene.* One of the genes carried on the X chromosome, for which the Y carries no allele. Sex-linked genes can occur singly in males, on the chromosome derived from the mother, and can occur as a pair in females.
- Somatic cell.* Any of the cells of the body except the germ cells.
- Species.* A group of actually or potentially interbreeding natural populations which is reproductively isolated from other such groups. The populations are the ethnic groups, the groups are the species.
- Synapsis.* The pairing, point by point, of homologous chromosomes before the maturation division of the gametes.
- Telophase.* The terminal stage of mitosis (or meiosis) during which a nucleus is re-formed around the chromosomes, and the latter uncoil and resume their elongated, threadlike appearance.
- Testosterone.* A male sex hormone produced by the testis and in small amounts by the cortex of the adrenal gland and probably also by the ovary.
- Tetrad.* The paired chromosomes of meiosis after each chromosome has duplicated itself and the pair is visibly four-stranded. Also, a quartet of cells formed by meiosis in a mother cell.
- Throwback.* See *atavism*.
- Trait.* See *character*.
- Valine unit.* A component of protein, as on the hemoglobin molecule.
- Variation.* The occurrence of differences in characters. *Discontinuous variation*, gradations of difference are perceptible in the phenotype. *Continuous variation*, gradations of difference are imperceptible in the phenotype.
- Viability.* The ability to survive.
- Virus.* A minute living organism which can be seen only by the electron microscope. Viruses are responsible for such diseases as smallpox, shingles, poliomyelitis, and numerous others.
- Vitamin.* An organic substance present in minute amounts in food-stuffs, necessary to normal metabolism, growth, and development.
- Zygote.* The fertilized ovum, the product of the union of the ovum and a spermatozoön.

Genetics in the U.S.S.R.: A Political "Science"

IN THE U.S.S.R. a dogmatic form of "genetics" has been officially decreed as the only politically acceptable version of that subject. The type of genetics described in the present volume is harassed and condemned by the Russians as "reactionary" or "capitalist Mendelian-Morganism." The matter is mentioned here only in order to answer the questions of those readers who have heard something of this development in Russia and who desire to have a plain statement of the facts.

This unfortunate subject can be dealt with only briefly here. There are two excellent books to which the reader may be referred for a more detailed account. These are Professor Conway Zirkle's *Death of a Science in Russia* (University of Pennsylvania Press, Philadelphia, 1949) and Sir Julian Huxley's *Heredity East and West* (Abelard-Schuman, New York, 1949).

The Russian version of genetics is known as "Michurinism" or "Lysenkoism," after the clever Russian gardener and plant breeder Ivan Vladimirovich Michurin (1855-1935) and Trofim Lysenko (1898-), plant breeder and the principal contemporary proponent of Michurinism. Both men were without scientific training. Lysenko has stated the essence of Michurinism in the following words: "The materialist theory of the evolution of living nature involves recognition of the necessity of hereditary transmission of individual characteristics acquired by the organism under the conditions of its life; it is unthinkable without recognition of the inheritance of acquired characters." As evidence in support of this Lysenko cites the example of "vernalization" of winter cereals. By treating the seeds with moisture at a low temperature cereals can be made to flower in winter that would otherwise flower in spring. This is not in the least a new discovery and has been known for a long time with respect to the modification of the flowering time of such seeds in a single generation, but Lysenko's claim that winter cereals can have their heredity permanently changed by such treatment so that they will behave like spring cereals in winter has not been substantiated by any scientist outside the U.S.S.R.

Lysenko has also claimed that heredity can be, as he calls it, "shattered." Shock treatment of various kinds, alterations in the environment, changes in metabolism can, according to Lysenko, permanently change

the heredity of the plant or organism. As a result of this "shattering" the heredity is said to become more plastic and malleable.

Is there any scientific evidence for these claims? The answer is: None at all. No scientist has been able to corroborate any of the claims made by the Lysenkoists. Indeed, all of these claims had long before the Michurinists, in one form or another, been demonstrated to be without foundation. Why, then, it will be asked, do the Russians insist upon maintaining this dogma of false ideas? Why have they destroyed so many geneticists who adhered to the scientifically demonstrated facts—men like Vavilov, Karpechenko, Levit, Ferry, Agol, Chetverikov, Serebrovsky, Ephroimson, Levitsky, and others?

The answer to this question appears to be that the Russians wanted a theory which maximized the role of environment in heredity and minimized the role of the genes—even to the extent of denying the very existence of genes!

It being a cardinal tenet of the communist creed that the uniqueness of man lies in his social rather than in his biological character, communist evolutionary thinking has tended to emphasize evolution through social rather than through biological changes. In placing the emphasis on social evolution, modification through environmental change becomes a principle applicable to all living things.

Theoretically there is nothing unsound in such a viewpoint. It becomes so only when it develops into an extreme environmentalism. And this is what has happened in the U.S.S.R.

Work of genuine interest, which is best approached with the tools of genetics, is often presented in a philosophical-political manner, together with claims that it is difficult to interpret, still less to substantiate. The customary settling of scientific disputes in an atmosphere of acrimony by political administrators, the dependence upon such administrators by scientists, has resulted in a rash of opportunism, in the lowering of the quality of research, and in a sterilization of brilliant minds in what was once a leading center of genetic activity.

Soviet genetics, for the most part then, is not so much a scientific discipline as an instrument of political expediency, a party-line ideology. To what purpose?

One of the principal purposes of the Russian government appears to be to lead their subjects to believe that hereditary differences between ethnic groups and between individuals are insignificant and can be easily permanently changed through modification of the environment. This idea seems particularly important to the Soviet leaders as a unifying principle in bringing the many different ethnic groups within the Soviet territories into the common fold. Cultural, physical, and mental differences are thus to be turned into cultural, physical, and mental likenesses. Men and women in the U.S.S.R. are to be not only politically equal but also biologically equal—by fiat of the state.

In his presidential address to the Eighth International Congress of Genetics held at Stockholm in July, 1948, the Nobel Prize-winner in genetics, Professor H. J. Muller, related how before the Russians called off the Seventh International Congress which was to be held in the U.S.S.R. in 1937 it had been proposed to proceed with it "provided all sessions and papers dealing with man were eliminated. At about the same time, the plans for a volume which was being prepared by leading geneticists of the world, designed to refute the Nazi racist doctrines, were abandoned. The clue was given when one of the men highest in the administration of applications of biological science, the head of agriculture in the Party, still asserted, even after having presided at most of the 1936 controversy on genetics, that evolution could not have occurred without the inheritance of acquired characteristics. On hearing this remark, the present writer asked this administrator whether this doctrine would not imply that the colonial, minority, and primitive peoples, those who had had less chance for mental and physical development, were not also genetically less advanced than the dominant ones. 'Ah, yes', he replied in confidential manner, and after some hesitation, 'Yes, we must admit that this is after all true. They are in fact inferior to *us* biologically in every respect, including their heredity. And that,' he added, 'is in fact the *official* doctrine.' The word 'official' here was his, although the italics are ours. And at this point we may interject the question, just who could be more official on the particular subject than this particular individual? The answer to this seems clear. 'But', continued our authority, 'after two or three generations of living under conditions of socialism, their *genes* would have so improved that *then* we would all be equal.' "*"

Still another reason why the political authorities would like to disseminate the view that acquired characters are inherited is that the genetically uninformed citizen is led to believe that by this means it becomes possible to alter the hereditary constitution of cultivated plants and domestic animals far more rapidly in desired directions than would be the case if it were necessary to select for mutations that have no adaptive relation to the conditions under which they arise. Vavilov was accused of trying to sabotage crop improvement because he said that it would probably take ten years or more to accomplish the results that Lysenko claimed could be achieved in a year or two.

The motivation for the promotion of Michurinism in the U.S.S.R. is clear. Whatever further motives may be involved it is clear that Michurinism is not science, and even though Lysenko has been appointed by his rulers as its prophet, neither the doctrine of Michurin nor the claims of Lysenko have any scientific merit whatever.

* H. J. Muller, "Genetics in the Scheme of Things," *Hereditas*, Supplementary Volume, 1949, p. 107.

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AUERBACH, CHARLOTTE, *Genetics in the Atomic Age*. Essential Books (Oxford University Press), New York, 1956.

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BARNETT, ANTHONY, *The Human Species*. Penguin Books Inc., Baltimore, 1957.

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